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Included in This Issue

OBSERVATIONS ON THE SEVERELY WOUNDED IN FORWARD FIELD HOSPITALS: WITH SPECIAL REFERENCE TO WOUND SHOCK *by*
John D. Stewart and Frank Warner

EVACUATION HOSPITAL EXPERIENCES WITH WAR WOUNDS AND INJURIES OF THE CHEST *by*
Paul W. Sanger

EARLY PULMONARY DECORTICATION IN THE TREATMENT OF POSTTRAUMATIC EMPYEMA *by*
Thomas H. Burford, Edward F. Parker and Paul C. Samson

THE IMMEDIATE AND LATE TREATMENT OF AN ARTERIOVENOUS FISTULA *by*
Emile Holman

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ANNALS OF SURGERY

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OBSERVATIONS ON THE SEVERELY WOUNDED IN FORWARD FIELD HOSPITALS: WITH SPECIAL REFERENCE TO WOUND SHOCK

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DURING the first nine months of 1944, the authors were privileged to carry out a study of a clinical nature on severely wounded soldiers admitted as nontransportable to forward field hospital units. The patients had undergone a screening process at the adjacent divisional clearing stations and in the shock tents of the field hospitals. Therefore, they comprised a selected group in urgent need of resuscitation and surgical treatment. The organization of the field hospital platoon functioning as a surgical hospital a few miles behind the front was such that a competent staff and adequate facilities were at hand for resuscitation, definitive surgical operation and postoperative care. In most instances, the surgical care of the patients was in the hands of surgical teams from an auxiliary surgical group. The patients were held from one to three weeks following operation until they could be safely evacuated. It was, therefore, possible to observe the patient's condition on admission in an essentially untreated state, after restorative therapeutic measures and periodically following operation. In addition, a second and smaller group of patients suffering from severe flak wounds was studied in a station hospital immediately after return from missions over enemy territory.

METHODS

To furnish facilities for making necessary biochemical measurements, an improvised laboratory mounted on a two and one-half ton truck (Fig. 1) was stationed at the door of the shock tent. The laboratory could be blacked out for night work and, on the whole, it proved to be a reasonably satisfactory substitute for the ordinary biochemical laboratory. The team was composed of two officers and two enlisted men, one of the latter being a biochemical technician and the other a truck driver and handy man. Since the laboratory was mobile, it could be moved to a point of greater activity when the flow of casualties fell off. Special laboratory apparatus and reagents essential for the study were obtained through the Theater Surgeon, and in various other ways.

Concentrations of hemoglobin and plasma protein, and red cell hematocrit were determined by the copper sulphate gravity method.[1] Normal values for the method, as obtained in a study of 42 front line soldiers with acute gonorrheal urethritis, are shown in Table II. The method proved entirely satisfactory under field conditions. The authors found that hematocrit values, as they obtained them with the method, agreed well with red cell volume as determined by centrifuging to a constant value. The ammonium and potassium oxalate mixture of Heller and Paul[2] was used as anticoagulant. Plasma volume was determined by Gregersen's method, using the blue dye T-1824 and the Decade Photometer devised by Nickerson[3] for field use. The simplicity, rapidity and ease of the method made it an invaluable aid, and the data obtained were of great value in controlling the intravenous therapy of hemorrhage and shock. In Table I are shown data from essentially normal ambulatory convalescent soldiers. Nonprotein nitrogen of the plasma was determined by tungstic acid precipitation of proteins followed by digestion and nesslerization.[4] Plasma chloride concentration was measured by the method of Wilson and Ball,[5] and total sulfonamide concentration by the method of Marshall and Litchfield.[6] Urinary ammonia was measured by digestion and nesslerization,[4] while titratable acidity was determined as described by Henderson and Palmer.[7] Analyses of the urine specimens were made daily on samples collected and stored immediately in the icebox in clean bottles containing thymol and toluol. Blood specimens were taken without stasis from vein or brachial artery. Samples were taken immediately after admission to the hospital and subsequently on a fasting basis.

FIG. 1.—Photograph showing the two and one-half ton truck used to furnish facilities for making the biochemical measurements.

CLINICAL MATERIAL AND MANAGEMENT

One hundred patients were studied in forward field hospital platoons, while a smaller group of 14 patients was seen in a station hospital receiving air combat crewmen returning from missions with severe flak wounds. All the patients had penetrating, perforating or lacerating wounds resulting from high-explosive missiles. In many instances, the wounds were multiple. It is to be emphasized that the patients were a selected group, chosen because of the extensiveness of their wounds and the presence or imminence of wound shock. All the patients were in an untreated state, practically speaking, when first seen after admission to the hospital. Splinting and bandaging, injection of morphine and tetanus toxoid, and in rare instances administration of plasma had occurred in forward aid stations or in the divisional clearing station. In many instances sulfanilamide had been applied to wounds and sulfanilamide tablets had been taken, but there were many exceptions.

All the patients were treated for shock and hemorrhage, and most of the patients were operated upon. In most instances, the nature of the wounds was such that definitive surgical operation was required, but in some cases of penetrating chest and head wounds and compound fractures, only such procedures as débridement, aspiration of hemothorax and splinting were carried out. Ether, frequently administered by endotracheal tube in a closed system, and intravenous sodium pentothal were the anesthetic agents used in most cases. None of the patients suffered from burns. However, for purposes of contrast, one burn case is considered in Table XXV but is in no other way included in the study. Plasma and blood were freely used in resuscitation and during operation, and to some extent postoperatively. Penicillin was given as a rule only in the treatment of infection but some of the patients during the latter part of the study were given 150,000 to 200,000 units daily as a routine measure during the first week after admission to hospital.

The severity of the wounds is attested by the high percentage of compound fractures, penetrating abdominal wounds and penetrating chest wounds, as shown in Tables IV, V and VI. Nevertheless, during the period of observation, varying from two or three days to four or five weeks, there were only 17 deaths out of the 100 patients admitted to the field hospitals, and two deaths among the 14 patients with flak wounds admitted to a station hospital. The difficulties of the terrain and the resultant problem in evacuation of the wounded are brought out in an analysis of the time factors, as shown in Table III. The average time from wounding to tagging and from tagging to admission to a field hospital are the significant figures. The average interval between admission to hospital and surgical operation, which was 3.5 hours, is a measure of the thoroughness of preoperative care, for at no time was there delay due to lack of facilities or surgical personnel. One of the patients in the group of 100 ground force casualties died after admission to hospital without responding sufficiently to restorative measures to permit operation.

On admission to the field hospital, the patients were triaged in the shock tent by the surgeon on call. As a rule, resuscitation was supervised by the surgeon who was to operate, and he it was who decided when the patient was in suitable condition for operation, sometimes a very difficult question. Postoperative care, likewise, was the responsibility of the operating surgeon, and, on the whole, this was conscientiously and skillfully attended to. For the most part, standard principles of war surgery were adhered to at operation. At celiotomy, exteriorization or resection of wounded small intestine was avoided wherever possible; wounded colon was exteriorized; liver wounds were usually packed with the end of a Penrose drain, though suture was sometimes performed; the damaged spleen was removed and the wounded kidney was resected or removed if required; suprapubic cystostomy was practiced in perforations of the bladder; thoracotomy was performed in cases of penetrating chest wounds in the presence of continued bleeding or uncertainty as to the extent of mediastinal or subdiaphragmatic trauma. In some instances upper abdominal surgery was done by the thoracic transdiaphragmatic approach. Large collections of blood in the pleural cavity were aspirated and aspiration or catheter drainage was the usual treatment for tension pneumothorax. Sucking wounds of the chest were closed as simply as possible by pressure dressing, or by suture of pleura and muscle or fascia with superficial drainage. Compound fractures were treated by débridement and plaster splinting, or by guillotine amputation if the extent of tissue loss so indicated.

Postoperative care was characterized by the free use of oxygen therapy, the administration of plasma, blood, glucose and saline intravenously, nasogastric continuous suction when needed, the intravenous administration of sodium sulfadiazine in most abdominal cases in addition to the application of sulfanilamide to the wounds. Nursing care was of a high quality and contributed greatly to the successful management of the cases.

In Table IX are noted pertinent data from the 17 instances in which death occurred among the 100 cases observed in field hospitals. Autopsy was performed in every instance, but only the gross pathologic findings can be reported. The incidence of clostridial infection, pulmonary thrombo-embolism, peritonitis, aspiration pneumonia, blast injury and irreparable shock are notable.

The variability in the clinical picture of wound shock was rather striking, and gave the impression that the usual concepts in the definition of traumatic shock are too rigid. The mental and emotional state of the patient, pain, blood pressure, pulse rate, sweating, nausea and vomiting, extent of filling of peripheral veins, skin temperature and plasma volume all were elements subject to considerable variation. It was clear that no single factor could be used in setting up a criterion for gauging the severity of shock, or the efficacy of restorative measures. Perhaps the most constant feature of the compensatory reactions in severe wound shock was the delicacy of the balance achieved and the rapidity with which a precarious adjustment could be improved or made worse. In illustration of this point the relationship between

systolic blood pressure and pulse rate should be mentioned. If these two factors are plotted together a rough inverse proportionality is detected, but exceptions are common, and low systolic blood pressure may be found with a normal pulse rate and normal or high blood pressure with a rapid pulse. Table XI contains pertinent data. It was frequently observed that patients brought into the shock tent without detectable peripheral blood pressure or pulse, after resting a few minutes under blankets in the head-down position and after receiving only an ounce or two of plasma, showed great improvement with normal or near-normal blood pressure and pulse.

PRESENTATION OF DATA AND DISCUSSION

From Table X, it is apparent that reduction in plasma volume was the rule in the severely wounded before replacement therapy. This was rapidly corrected through natural adjustments and by parenteral fluid therapy. The data, weight and surface area could not be obtained in these patients, so there is only the unreliable figure of last-remembered weight to refer plasma volume to. There were enough cases under examination, however, so that average values can be used to set up standard figures. The volume of the whole blood was proportionately more depleted than that of the plasma, as analysis of the changes in hematocrit and plasma protein concentration will show. If the average values for plasma volume, plasma protein concentration and hematocrit during the first 24 hours are compared with those for the second period (one to four days), it is evident that an increase in blood volume was effected despite a reduction in concentration of both hemoglobin and plasma protein. If total circulating hemoglobin and plasma protein are computed at the two periods, it can be seen that initial deficits of hemoglobin are relatively greater than deficits of plasma protein and are less easily corrected. The validity of this observation is somewhat depreciated by the uncertain effects of plasma infusion and blood transfusions during the periods considered. With respect to administration of plasma and whole blood, it was repeatedly noted that quantitative changes do not result from such therapy. These observations lead to provocative questions, but one obvious explanation is that depleted storehouses for plasma protein and hemoglobin have a high priority during restorative therapy. The data shown in Tables XVI to XXIV, inclusive, bear on this point.

Hemoconcentration, either of red cells or plasma protein, was conspicuously absent even in the cases seen quite early after severe wounding. The one case of severe burn (Table XXV) is inserted for contrast. All these patients were gravely wounded, some were in a state of irreparable traumatic shock, and yet in every instance the initial measurements before treatment disclosed either normal or, much more commonly, reduced hemoglobin and plasma protein values. Loss of blood into tissues, body cavities and to the outside dominated the picture and clearly afforded the clue to proper resuscitation. In considering average plasma protein values for the various periods, as seen in Table X, at no time was the average below the range of

TABLE I

PLASMA VOLUME DETERMINATIONS; 53 MEASUREMENTS ON 29 AMBULATORY CONVALESCENT SOLDIERS

	Cc.	Cc./Kg.
Average	3290	45
High	4650	57
Low	2450	38

TABLE II

VALUES FOR HEMATOCRIT AND PLASMA PROTEIN CONCENTRATION BY CU SO_4 GRAVITY METHOD AS DETERMINED ON 42 FRONT-LINE SOLDIERS WITH GONORRHEA

	Hematocrit %	Plasma Protein Gm/100/cc.
Average	43.3	7.0
High	50.5	8.0
Low	37.5	6.2

TABLE III

TIME RELATIONSHIPS IN MEDICAL CARE AFTER SEVERE WOUNDING; 100 GROUND FORCE CASUALTIES ADMITTED TO FORWARD FIELD HOSPITAL PLATOONS

	Average Hours	Minimum Hours	Maximum Hours
Time, wounding to tagging	1.80	0.10	18.00
Time, wounding to admission to hospital	4.90	0.60	25.20
Time, wounding to operation	8.45	2.75	26.00

TABLE IV

COMPOUND FRACTURES AS RECEIVED BY 55 OUT OF 100 GROUND FORCE CASUALTIES WOUNDED BY HIGH EXPLOSIVE MISSILES INCLUDING "TRAUMATIC AMPUTATION" IN 17

Femur	13
Tibia	12
Feet	9
Fibula	7
Ulna	5
Humerus	4
Ribs	4
Skull	4
Spine	3
Radius	3
Hand	3
Pelvis	2
Scapula	2
Clavicle	1

TABLE V

PENETRATING ABDOMINAL WOUNDS AS RECEIVED BY 48 OUT OF 100 GROUND FORCE CASUALTIES WOUNDED BY HIGH EXPLOSIVE MISSILES; VISCERA WOUNDED

Colon	23
Small intestines	21
Liver	14
Stomach	10
Spleen	7
Kidney	6
Duodenum	4
Rectum	2
Bladder	1

TABLE VI

WOUNDS AS RECEIVED BY 100 GROUND FORCE CASUALTIES FROM HIGH EXPLOSIVE MISSILES, SEEN IN FORWARD FIELD HOSPITAL PLATOONS

Compound fractures	55
Penetrating abdominal wound	48
Penetrating thoracic wound	32
Penetrating abdominothoracic or abdominal and thoracic	19

TABLE VII

WOUNDING AGENT IN 100 GROUND FORCE CAUSALTIES FROM HIGH EXPLOSIVE MISSILES

Shell fire	80
Mine	8
Grenade	6
Bullet	5
Aerial bomb	1

TABLE VIII

PLASMA AND BLOOD TRANSFUSIONS DURING FIRST 24 HOURS AFTER ADMISSION TO FORWARD HOSPITAL FOLLOWING SEVERE WOUNDING—87 PATIENTS. IN EIGHT CASES NEITHER BLOOD NOR PLASMA WAS GIVEN DURING THE PERIOD.

Plasma Therapy			Blood Therapy		
Average	High	Patients Receiving None	Average	High	Patients Receiving None
720 cc.	2500 cc.	10	1130 cc.	4000 cc.	28

TABLE IX

DEATHS OCCURRING IN 100 SEVERELY WOUNDED GROUND FORCE CASUALTIES ADMITTED TO FORWARD FIELD HOSPITALS

Case Number	Survival Period—Days	Autopsy Findings and Clinical Diagnosis
6	2	Abdominothoracic wounds, perforation of the esophagus; shock and hemorrhage
8	1	Perforating abdominal wound, compound fracture of femur; severe shock
9	1	Blast injury both lungs, compound fracture of clavicle, lacerations of chest wall
10	4	Penetrating wounds of abdomen and brain, traumatic amputation of foot massive, bilateral pulmonary infarcts
16	36	"Traumatic amputation" of thigh, clostridial myositis, massive empyema
21	4	Penetrating abdominal wound, perforation of colon, ileum, kidney; pulmonary embolism
25	1	Perforating wound of mediastinum, aspiration of vomitus during anesthesia; died at operation
26	1	Penetrating wound of abdomen, perforation of ileum, colon, rectosigmoid; aspiration of vomitus during anesthesia; acute dilation of stomach
27	3	Penetrating abdominal wound, perforation of liver, stomach, jejunum; resection of jejunum; fibrinous peritonitis, edema of lungs
28	12	Penetrating abdominal wound, perforation of colon and jejunum, "traumatic amputation" of thigh, perforating wound of buttocks; clostridial myositis, bronchopneumonia
32	5	Penetrating thoraco-abdominal wounds, perforation of colon, small intestine; "retroperitoneal cellulitis with gas formation"
33	1	Penetrating thoraco-abdominal wound, abdominal evisceration; severe shock
38	1	Penetrating wounds of chest, abdomen, thigh, perforation of colon, feculent peritonitis
39	13	Penetrating wound of chest, laceration of thigh; retroperitoneal infection; "blast injury" of heart and lungs; multiple pulmonary emboli
61	1	Penetrating abdomino-thoracic wounds, perforation of liver, spleen, stomach; severe shock; death at operation
65	1	Multiple penetrating wounds of abdomen and chest; severe shock; died before operation
111	4	Penetrating wounds of abdomen, perforation of colon, acute dilatation of stomach, bilateral pneumonia

TABLE X

PLASMA PROTEIN AND HEMATOCRIT VALUES AT VARYING INTERVALS AFTER SEVERE WOUNDING; INITIAL DETERMINATIONS MADE BEFORE RESUSCITATION—87 PATIENTS

Interval Days, Inclusive	Plasma Protein, Gm./100 Cc.				Hematocrit, %				Plasma Volume, Cc.			
	Avg.	High	Low	No. of Observations	Avg.	High	Low	No. of Observations	Avg.	High	Low	No. of Observations
Initial	6.47	7.5	5.1	71	37.5	60.0	17.0	86	2950	4250	1030	84
1–4	6.30	7.7	4.6	114	34.5	48.5	23.0	102	3470	4650	2700	44
5–8	6.32	7.7	4.5	50	33.6	46.0	19.5	50	3290	3750	2850	7
9–12	6.41	7.4	5.1	33	36.3	48.2	26.5	33	3480	4450	2850	12
13 plus	6.55	8.2	4.8	9	39.8	50.5	30.5	22	3400	4100	2900	7

TABLE XI

DATA ON WOUNDED GROUPED ACCORDING TO BLOOD PRESSURE; MEASUREMENTS ON ADMISSION TO HOSPITAL BEFORE RESUSCITATION

		Blood Pressure				Plasma Volume Cc.	Plasma Protein Gm./100Cc.	Hematocrit %	Infusions Before and During Operation	
		Syst.	Diast.	Pulse P.	Pulse Rate				Plasma Cc.	Blood Cc.
Systolic B.P. zero	Average					2490	6.12	37.9	620	1340
12 cases (5 deaths)	High				140	3300	7.30	56.0	1250	4000
	Low				120	1030	5.10	31.4	250	0
Systolic B.P. less than	Average	85	47	38	122	2890	6.34	36.8	840	1220
100	High	98	70	88	160	4400	7.50	60.0	2000	3000
39 cases (9 deaths)	Low	38	20	18	80	1250	5.50	17.0	0	0
Systolic B.P. 100 or	Average	120	69	52	102	2960	6.43	38.4	620	920
over	High	154	104	108	148	4250	7.50	49.7	2500	2000
36 cases (4 deaths)	Low	102	30	27	80	1820	5.60	26.0	200	0

TABLE XII

DATA ON WOUNDED GROUPED ACCORDING TO PLASMA VOLUME; MEASUREMENTS ON ADMISSION AND BEFORE RESUSCITATION

		Blood Pressure				Plasma Volume Cc.	Plasma Protein Gm.%	Hematocrit %	Infusions Before and During Operation	
		Syst.	Diast.	Pulse P.	Pulse Rate				Plasma	Blood
Cases with PV of	Average	99	58	41	112	2200	6.25	37.2	640	1090
2700 or below	High	140	104	72	156	2700	7.2	56.0	1500	4000
(33 cases, 10 deaths)	Low	0	0	0	80	1030	5.1	17.0	170	250
PV of 2701 to 3200	Average	89	55	44	112	2950	6.43	36.6	860	1070
inclusive	High	154	80	108	148	3200	7.5	45.0	2500	2500
(30 cases, 6 deaths)	Low	0	0	0	84	2700	5.5	26.0	200	350
PV of 3201 or above	Average	105	62	42	108	3640	6.45	38.4	660	1130
(24 cases, 2 deaths)	High	138	84	88	140	4400	7.5	60.0	1125	3000
	Low	0	0	0	74	3300	5.6	28.0	200	500

TABLE XIII

DATA ON WOUNDED GROUPED ACCORDING TO HEMATOCRIT (VC); MEASUREMENTS ON ADMISSION AND BEFORE RESUSCITATION

		Blood Pressure				Plasma Volume Cc.	Plasma Protein Gm.%	Vc. %	Infusions Before and During Operation	
		Syst.	Diast.	Pulse P.	Pulse Rate				Plasma Cc.	Blood Cc.
Cases with Vc 35.0	Average	95	53	44	116	2815	5.99	31.0	950	1130
or below	High	154	80	88	156	3500	6.8	35.0	2500	2000
(24 cases, 3 deaths)	Low	0	0	0	74	2050	5.5	17.0	250	350
Cases with Vc 35.1 to	Average	96	59	40	112	2900	6.27	37.7	670	1050
40.0 inclusive	High	138	104	88	160	4400	7.5	40.0	2000	3000
(39 cases, 9 deaths)	Low	0	0	0	80	1030	5.1	35.4	200	250
Cases with Vc 40.1	Average	103	62	44	109	2864	6.63	44.5	560	1070
or higher	High	140	90	108	160	4100	7.5	56.0	1000	2000
(24 cases, 6 deaths)	Low	0	0	0	80	1820	5.6	40.5	100	500

TABLE XIV

MEASUREMENTS OF PLASMA NONPROTEIN NITROGEN CONCENTRATIONS AT VARYING INTERVALS AFTER SEVERE WOUNDINGS IN 95 GROUND FORCE CASUALTIES TREATED IN FORWARD FIELD HOSPITAL PLATOONS; VALUES EXPRESSED IN MG./100 CC.

	1st Day	2-3 Days	4-6 Days	7-12 Days	13-21 Days
Average	30.8	37.6	44.1	50.2	30.9
High	64.5	102.0	115.0	125.0	94.0
Low	14.8	19.8	20.4	18.2	21.4
No. of observations	67	55	31	23	14

TABLE XV

WHOLE BLOOD TOTAL SULFONAMIDE CONCENTRATION IN 89 GROUND FORCE BATTLE CASUALTIES AT VARYING INTERVALS AFTER SEVERE WOUNDING; ALL PATIENTS HAD RECEIVED SULFONAMIDE TOPICALLY, ORALLY, OR PARENTERALLY; VALUES EXPRESSED IN MG./100 CC.

	Period, Days After Wounding					
	1	2	3	4–6	7–10	11–21
Average value, cases with positive tests	3.6	7.5	7.8	8.6	6.7	5.1
Highest value for period	6.5	20.0	25.0	25.0	15.0	13.2
Cases with values above 12.0 mg. %	0	4	7	8	3	1
Number of cases with positive tests	5	24	37	43	36	35
Number of cases with negative tests	53	12	6	4	11	24
Total number of cases	58	36	43	47	47	59

TABLE XVI

CASE 31: SHELL FRAGMENT WOUND, PENETRATING RIGHT HEMOTHORAX, DIAPHRAGM AND KIDNEY; LACERATION OF LIVER. CELIOTOMY

			Blood Values							I. V. Therapy		
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid
2/4	1100	Wounded										
	1115	Tagged										
	1145	Admitted to field hospital								150		
	1209	Blood studies	6.7	39.1	13.2	2900	26.8		0			
	1630	Operation								500	500	1000
2/5		Severe chill following transfusion									500	
2/6	0700	Blood studies	6.5	44.0	14.7				8.7			1300
2/8	0713	Blood studies	6.2	42.0	14.2		38.2		4.4			1000
2/10	0715	Blood studies	6.3	38.5	13.0				2.7			
2/12	0730	Blood studies	7.3	41.0	13.8				8.5			
2/14											500	
2/15	0730	Blood studies	7.2	39.5	13.3				4.0			
2/17	0747	Blood studies	6.8	39.5	13.4	3200	29.3		5.3			
2/19	0720	Blood studies	6.7	38.5	13.0				4.2			
2/21	0745	Blood studies	6.7	37.5	12.6				0			
2/23	0740	Blood studies	6.7	38.5	13.0				3.1			
2/25	0731	Blood studies Evacuated	6.3	38.0	12.9	3150	21.5	92.5	5.5			

Patient's weight—165 lb. (August, 1943). Height—66 inches.

On admission to hospital patient's condition was fairly good: B. P.—102/75, P.—84.

Chemotherapy: 4 Cm. sulfanilamide orally after wounding, 5.0 Gm. sulfanilamide into wounds at operation, 75.5 Gm. sulfadiazine or sod. sulfadiazine orally or I.V. through 2/25.

PP equals plasma protein in Gm. per 100 cc.; Vc equals red cell hematocrit in percentage; Hb equals hemoglobin in Gm. per 100 cc.; PV equals plasma volume in cc.; NPN equals plasma nonprotein nitrogen in mg. per 100 cc.; Cl equals plasma chloride in milli-equivalents per liter; sulfa equals blood total sulfonamide in mg. per 100 cc.; I.V. therapy equals intravenous infusions in cc. of plasma, blood or 5% glucose in saline solution.

TABLE XVII

CASE 30: SHELL FRAGMENT WOUND, PENETRATING LUNG, DIAPHRAGM, SPLEEN. THORACOTOMY, CELIOTOMY, SPLENECTOMY.

			Blood Values							I. V. Therapy		
Date	Time	Remarks	PP	VC	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid
2/11	1330	Wounded										
	1340	Tagged										
	1410	Admitted to field hospital								400		
	1448	Blood studies	6.0	34.0	11.4	2400	22.8		0			
	1730	Operation								150	1500	
2/12										500		3000
2/13	0730	Blood studies	6.0	37.5	12.4				4.8			
2/14											500	
2/15	0709	Blood studies	5.6	37.0	12.3	3300	25.0		4.4		500	
2/17	0730	Blood studies	6.5	40.5	13.7				2.6			
2/19	0715	Blood studies	6.5	40.5	13.6				5.8		500	
2/21	0730	Blood studies	6.7	43.5	14.7				6.7			
2/22	0717	Blood studies	7.0	45.5	15.4		29.1	96.7	tr			
2/24	0715	Blood studies	6.8	44.0	14.8				6.3			
2/25		Evacuated										

Patient's weight 140 lbs. (February, 1943). Height—67 inches.

On admission to hospital B.P. was 118/80, skin was ashen and cold. During operation B.P. dropped to 70/52. P. 144.

Chemotherapy: 37.5 Gm. sod. sulfadiazine or sulfadiazine given I.V. or P.O. through 2/24. (See Table XVI for legend)

TABLE XVIII

CASE 39: SHELL FRAGMENT WOUND, PERFORATION OF THORAX AND RETROPERITONEAL REGION, LACERATIONS RIGHT THIGH; TRAUMATIC SHOCK; THORACOTOMY AND DÉBRIDEMENT; DEATH

			Blood Values							I. V. Therapy		
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid
2/14	1740	Wounded										
	1815	Tagged								750		
	2115	Admitted to field hospital										
	2127	Blood studies	5.6	35.4	12.0	2700	29.2		0			
	2300	Operation								500	500	
2/15												2000
2/16	0730	Blood studies	5.5	32.5	11.0				tr.			
2/17	0712	Blood studies	5.7	32.0	10.8	3100	37.5		1.8			1200
2/19	0715	Blood studies	4.8	24.5	8.2				11.1	500	500	1000
2/21	0730	Blood studies	4.5	34.0	11.5				9.3		500	
2/23	0700	Blood studies	5.5	34.5	11.7				8.4			
2/25	0706	Blood studies	5.6	38.5	13.2	3300	25.1		7.8	250	500	
2/27	0730	Blood studies	4.8	47.5	16.1			95.8	5.9	250	1000	2000
	1535	Death										

On admission to hospital, the patient's B.P. was 0/0, pulse not palpable.

Sulfonamide therapy: 2.5 Gm. sulfanilamide taken orally after wounding; 5.0 Gm. sod. sulfadiazine given I.V. daily.

Autopsy: Severe retroperitoneal sepsis, infected wounds of left thigh, multiple pulmonary infarcts, blast injury of heart and lungs, hemothorax, rt.

Weight: 158 lbs. (April, 1943). Height—67 inches. (See Table XVI for legend)

TABLE XIX

CASE 51: PERFORATING GUNSHOT WOUND OF UPPER ABDOMEN THROUGH LIVER, COLON; TRAUMATIC SHOCK; CELIOTOMY, SUTURE OF LIVER, EXTERIORIZATION OF COLON

			Blood Values							I. V. Therapy		
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid
4/20	0600	Wounded										
	0700	Tagged								750		
	0820	Admitted to field hospital										
	0844	Blood studies	6.5	35.0	11.9	2950	23.5	101.0	0			
	1130	Operation									1500	1000
												1000
												2000
4/21	0730	Blood studies	6.3	45.5	15.5				11.4			
4/22	0726	Blood studies	6.3	35.0	11.8	3750	29.6	104.4	5.1	250		2000
												2000
4/24	0715	Blood studies	7.6	30.0	10.3				7.9			1000
4/26	0740	Blood studies	6.3	31.0	10.4				4.8			
4/28	0715	Blood studies	6.3	33.5	11.2				4.2			
4/30	0715	Blood studies	6.5	29.5	10.1				7.5			
5/2	0735	Blood studies	7.2	33.0	11.1				3.6			
5/3	0735	Blood studies	7.3	34.5	11.3				6.1			
5/5	0730	Blood studies	7.0	35.0	11.8				10.0			
5/7	0735	Blood studies	6.7	33.0	11.1				8.3			
5/9	0735	Blood studies Evacuated	6.7	34.5	11.6				9.4			

On admission to field hospital, B.P. was 124/72 (after 750 cc. plasma), skin was moist, mind was clear.

Sulfonamide therapy: 7.5 Gm. sulfanilamide into wounds at operation; 70.5 Gm. sodium sulfadiazine given I.V. in daily doses 4/21 to 5/9.

Weight—175 lbs. (September, 1943). Height—68 inches. (See Table XVI for legend)

TABLE XX

CASE 27: SHELL FRAGMENT WOUND, PENETRATING ABDOMEN, WITH PERFORATION OF STOMACH, JEJUNUM AND LIVER. CELIOTOMY RESECTION OF JEJUNUM, SUTURE OF STOMACH AND LIVER. POSTOPERATIVE OLIGURIA; DEATH

			Blood Values							I. V. Therapy			
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid	Urine Volume
2/21	1500	Wounded											
	1525	Tagged											
	1710	Admitted to field hospital								750			
	1738	Blood studies	6.8	41.5	14.0	3350	22.2	97.6	tr.				
	1825	Operation								1000	2000		
2/22	0740	Blood studies	5.0	42.0	14.2				18.8			2000	250
												2000	
2/23	0657	Blood studies	6.3	40.5	13.7	3600	88.2	99.3	19.4			1000	550
	2330	Blood studies											
2/24	0740	Blood studies	6.0	39.5	13.3		94.0	107.0	11.7	250	500	2000	?
	1600	Venesection, 500 cc.											
	1910	Death											

On admission to hospital, patient's B.P. was 130/75, P. 140; occasional vomiting.

During operation B.P. was unstable and at one time dropped to 70/40.

Sulfonamide therapy: 2.5 Gm. sulfanilamide taken by mouth after wounding, 5 Gm. sod. sulfathiazole given I.V. after operation, 10 Gm. sulfanilamide put into peritoneal cavity at operation.

Autopsy: Engorgement of abdominal viscera, fibrinous peritonitis, edema of both lungs, serosanguineous bilateral pleural effusion, cloudy swelling of both kidneys.

Weight—170 lbs. (February, 1943). Height—73 inches. (See Table XVI for legend)

TABLE XXI

CASE 66: SHELL FRAGMENT WOUND, PENETRATING CHEST, ABDOMEN; PERFORATION OF COLON, SPLEEN, KIDNEY, SMALL INTESTINES; SPLENECTOMY; TRAUMATIC SHOCK; POSTOPERATIVE OLIGURIA AND RENAL INSUFFICIENCY; RECOVERY

			Blood Values							I. V. Therapy			
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid	Urine Volume
5/3	0635	Wounded											
	0640	Tagged											
	0925	Admitted to field hospital B.P. 0/0, P. 140								250			
	0931	Blood studies	6.5	35.0	12.0	2150	33	101.9	0				
										250	1000		
	1300	Operation									1000	1000	
5/4	0735	Blood studies	7.2	48.5	16.5				18.1			3000	
												1000	160
5/5	0648	Blood studies	6.1	44.0	14.9	3200	102	100.2	25.0			2000	700
5/6	0735	Blood studies	6.3	32.5	11.0		115		25.0			1000	1800?
5/7	0725	Blood studies	7.0	32.0	10.6		116		21.7		500	1700	700?
5/8	0700	Blood studies	7.0	39.5	13.2		124	95.4	10.7			3500	2245
5/9	0715	Blood studies	7.7	40.0	13.4		118	102.3	5.4		500	1000	2375
5/10	0730	Blood studies					125	102.2			500	1000	?
5/11	0745	Blood studies	7.7	46.0	15.5				3.5			1000	?
5/12	0759	Blood studies	7.0	39.0	13.2	2850	94	101.9	tr.				?
5/13		Evacuated											

Weight—140 lbs. (Sept., 1944). Height—64 inches.

On admission to field hospital B.P. 0/0, P (carotid) 140, skin cyanotic but dry, responded slowly but intelligently to questioning. Normal wt. 120 lbs., last wt. Sept., 1943, 140 lbs., height, 64 inches.

Sulfonamide therapy: sulfanilamide in wound dressing, no tablets taken; 20 Gm. to peritoneum at operation, 5 Gm. sod. sulfadiazine I.V. at conclusion of operation.

Urine: 5/4: sp. gr. 1.014, albumin plus plus, loaded with red cells, many granular casts.
5/7: sp. gr. 1.014, albumin plus plus plus, acid, loaded with red cells.
5/9: yellow, acid, sp. gr. 1.015, albumin plus plus plus, occasional red blood cell, occ. white blood cell.

Blood pressure: 5/7—165/82, 5/8—200/90.

Special medication: 5/5: 150 cc. 10% $MgSO_4$ plus 500 cc. 0.5% KCl I.V.
5/6: KCl 6.25 Gm. IV., $MgSO_4$ 10 Gm. I.M. and 20 Gm. I.V. (See Table XVI for legend)

TABLE XXII

CASE 45: SHELL FRAGMENT WOUND, COMPOUND FRACTURE OF FEMUR AND AVULSION OF THIGH; PROBABLE RENAL DAMAGE

			Blood Values							I.V. Therapy			
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid	Urine Volume
4/24	0545	Wounded											
	0630	Tagged											
	0700 / 1300									2500			
	1530	Admitted to field hospital											
	1536	Blood studies	6.6	26.0	8.9	2900	47	97.2	3.0				
											1500	250	
	1910	Blood studies	7.0	32.5	11.0								
	1920	Operation started									1000	500	
	2130	Blood studies	6.0	30.0	10.2							500	
4/25	0730	Blood studies	6.3	27.1	9.2				13.6	500		1000	
4/26	0706	Blood studies	5.6	23.5	8.0	3050	75	94.7	12.8	500		750	1200
4/27	1000	Blood studies					83				1500	1000	2450
4/28	0715	Blood studies	6.2	31.0	10.3		85		4.9		1000		1990
4/29	0715	Blood studies	6.5	38.0	12.9		91	100.7	4.6		1000		1700
4/30	0715	Blood studies	6.7	43.0	14.3		96		2.9				1700
5/1	1000	Blood studies	6.8	44.0	14.9		100		2.0		500		2650
5/2													2350
5/3	0725	Blood studies	6.7	41.5	14.0		66		tr.				2490
5/4	0710	Blood studies Evacuated	6.8	42.0	14.2	3450	62	106.6	3.8				

On admission to field hospital: B.P. 124/44, P 104, BP remained up.

Sulfonamide therapy: 4.0 Gm. sulfanilamide orally after wounding; 15.0 Gm. in wound at operation.

4/26 Urine: sp.gr. 1.012, PH 8.0, albumin plus, epithelial cells and coarsely granular casts.

4/28 Urine: sp.gr. 1.010, PH 7.0, albumin plus, occ. W.B.C.

4/30 Urine: sp.gr. 1.010, PH 4.0, albumin plus, occ. W.B.C.

Patient's weight—145 lbs. for past 3-4 years; last weighed Sept., '43. Height—67.5 inches.

(See Table XVI for legend)

TABLE XXIII

CASE 16: SHELL FRAGMENT WOUND OF BOTH THIGHS, COMPOUND FRACTURE OF LEFT FEMUR; AMPUTATION LEFT THIGH, CLOSTRIDIAL MYOSITIS OF THIGH PENICILLIN THERAPY

			Blood Values							I.V. Therapy	
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood
1/22	0430	Wounded									
	0530	Tagged									
	0710	Admitted to field hospital, B.P. 126/74, P. 156								500	1000
	1030	Operation									2000
1/25	0600	Peripheral vascular collapse, clostridial myositis									
	1700	Blood studies	5.6	32.0	11.0				1.8		
1/28	1000	Blood studies. Extensive wound edema	5.0	53.0	17.8					1000	
2/2	0700	Blood studies	4.6	43.5	14.8	1540	33.9		0	500	500
2/4	0737	Blood studies	5.5	35.0	11.8	2300	24.4		0		
2/7	0700	Blood studies	4.6	29.1	10.0				0		
2/9	0815	Blood studies	5.5	36.0	12.2				tr.		
2/10	0730	Blood studies	5.1	34.5	11.8				1.5		
2/13	0745	Blood studies	6.0	42.0	14.2				tr.		500
2/16	0800	Blood studies	6.0	45.0	15.2				0	500	500
2/19	0720	Blood studies	6.2	44.5	15.1				0		500
2/22	0750	Blood studies	6.0	47.5	16.2				0		
2/25	0755	Blood studies	6.5	46.8	15.8				0		
2/26	0638	Blood studies. Evacuated	6.3	45.5	15.3	2850	20.0	87.0	0		

Development of clostridial myositis in thigh amputation stump heralded by sudden peripheral vascular collapse; edema and muscle necrosis followed.

Chemotherapy: 5 Gm. sulfanilamide taken by mouth after wounding. Penicillin (200,000 units per day), sulfathiazole (4 Gm. per day) from 28 January to 21 February. Gas antitoxin given at onset of infection.

Outcome: Condition improved steadily until after evacuation; death subsequently from undetected extensive empyema.

Weight—136 lbs. Height—67 inches. (See Table XVI for legend)

TABLE XXIV

CASE 28: SHELL FRAGMENT WOUNDS, MULTIPLE, PENETRATING ABDOMEN, JEJUNUM. COLON, BUTTOCKS; AMPUTATION OF LEFT THIGH; TRAUMATIC SHOCK; CLOSTRIDIAL MYOSITIS IN THIGH WOUND; DEATH DESPITE PENICILLIN THERAPY

			Blood Values							I. V. Therapy		
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid
2/12	0700	Wounded										
	0845	Tagged										
	1910	Admitted to field hospital; B.P. 98/60, P. 128								1500		
	1931	Blood studies	6.3	28.5	9.8	3200	34.9		0	500	1000	
2/13	0300	Operation. Clostridial myositis of thigh suspected									1000	
		Severe postop. hemorrhage from thigh stump									500	1000
2/14	0730	Blood studies	6.7	30.5	10.3		30.0		12.6	250	750	2000
2/15											500	4000
2/16	0752	Blood studies	5.6	32.0	10.8	3125	30.0		tr.			2000
2/17												2000
2/18	0740	Blood studies	5.0	34.5	11.8				tr.			1000
2/20	0730	Blood studies. Extensive edema of trunk	4.8	31.5	10.7				tr.			
2/22	0730	Blood studies	4.8	36.5	12.3				4.0			
2/24	0530	Died										

Clostridial myositis of amputation stump suspected initially at operation from odor and appearance of wound. Penicillin therapy started (120,000 units daily) and continued until death. Sod. sulfadiazine started I.V. 2/14 at rate of 2.5 Gm. daily.

Autopsy: Anaerobic infection (probably clostridial myositis) of buttocks and thigh amputation stump; partial pulmonary atelectasis and early pneumonia of right lower lobe. Peritoneal cavity clean.

Weight—140 lbs. (February, 1943). (See Table XVI for legend)

TABLE XXV

CASE 64: BURNS, GENERALIZED, SECOND AND THIRD DEGREE, FROM EXPLOSION OF FLAME THROWER. DEATH

			Blood Values							I. V. Therapy		
Date	Time	Remarks	PP	Vc	Hb	PV	NPN	Cl	Sulfa	Plasma	Blood	Other Fluid
5/11	0920	Burned										
	0930	Tagged										
	1220	Admitted to field hospital								1375		
	1252	Blood studies	7.3	59.0	20.0	2250	35.1	98.6	0			
	1330	Surgical dressing								2150		1000
5/12	0700	Tracheotomy										
	1000	Death										

On admission to hospital the B.P was 128/98, P. 112, respirations 9. The patient answered questions intelligently and quickly but spoke in a whisper. The blood at venepuncture was dark and viscous.

Autopsy: Diffuse capillo-venous engorgement of pulmonary tissue, acute tracheobronchitis and laryngitis with fibrinous exudate.

Weight—190 lbs. (3 months ago). Height—70 inches. (See Table XVI for legend)

TABLE XXVI

OBSERVATIONS ON THE SPECIFIC GRAVITY OF FRESHLY VOIDED URINE OF SEVERELY WOUNDED SOLDIERS

	1st Day	2nd Day	3rd Day	4th Day
Number of samples voided	108	84	58	26
Number of patients	20	19	15	6
Average specific gravity	1.021	1.020	1.020	1.020
Highest specific gravity	1.033	1.032	1.034	1.030
Lowest specific gravity	1.001	1.003	1.004	1.007

TABLE XXVII

OBSERVATIONS ON THE REACTION OF FRESHLY VOIDED URINE (USING NITROZINE PAPER) OF SEVERELY WOUNDED SOLDIERS; NONE HAD $NAHCO_3$ THERAPY

Day	1st	2nd	3rd	4th	5th	6th
Number of patients	7	14	12	11	7	4
Number of samples	7	64	69	59	24	14
Acid reaction	5	25	26	41	16	10
Neutral reaction	0	9	13	10	6	4
Alkaline reaction	2	30	30	8	2	0

TABLE XXVIII

DETERMINATION OF URINE VOLUME, AMMONIA, AND TITRATABLE ACID AT VARYING INTERVALS IN SEVERELY WOUNDED SOLDIERS; 24-HOUR TOTAL VALUES TABULATED

	1st Day (14 Patients)			2nd Day (12 Patients)			3rd Day (10 Patients)			4th Day (5 Patients)			5th Day (2 Patients)		
	Vol. Cc.	NH_4 mEq.	Tit.Ac. mEq.	Vol. Cc.	NH_4 mEq.	Tit.Ac. mEq.	Vol. Cc.	NH_4 mEq.	Tit.Ac. mEq.	Vol. Cc.	NH_4 mEq.	Tit.Ac. mEq.	Vol. Cc.	NH_4 mEq.	Tit.Ac. mEq.
Avg. value	1690	27.8	15.8	1614	43.0	22.1	1240	56.3	22.3	1400	53.3	21.8	1185	45.1	10.0
Highest value	6100	49.1	35.0	3460	82.8	43.7	2160	107.1	53.5	1885	88.0	34.7	1320	48.8	10.5
Lowest value	400	7.7	0	550	11.0	0	680	0	0	720	1.6	12.6	1055	41.4	9.6

TABLE XXIX

DETERMINATION OF URINE VOLUME, AMMONIA AND TITRATABLE ACID AT VARYING INTERVALS IN SEVERELY WOUNDED SOLDIERS, EACH GIVEN A TOTAL OF 40 GM. $NAHCO_3$ INTRAVENOUSLY DURING 1ST AND 2ND DAYS; 24-HOUR TOTAL VALUES TABULATED—5 PATIENTS

	1st Day			2nd Day			3rd Day		
	Vol. Cc.	NH_4 mEq.	Tit.Ac. mEq.	Vol. Cc.	NH_4 mEq.	Tit.Ac. mEq.	Vol. Cc.	NH_4 mEq.	Tit.Ac. mEq.
Average value	1845	17.3	7.0	1125	13.8	3.0	1090	35.2	13.5
Highest value	2745	39.1	23.5	1675	18.2	6.7	1425	57.4	35.3
Lowest value	1045	7.8	0	400	4.8	0	750	23.8	0.3

normal by the method used. Individual readings, however, were frequently below normal, the lowest being 4.5 Gm./100 cc. In only one instance, and in that more than 12 days after wounding, was the highest plasma protein concentration above the normal range. Much the same holds for hemoglobin, though the average values during the fourth to twelfth days were below the range of normal. In other words, during convalescence and despite parenteral therapy, mild hypoproteinemia and definite anemia were the rule.

In evaluating the state of wound shock in these patients, several criteria may be tested. The systolic blood pressure, pulse pressure and pulse rate are pertinent, as are plasma volume, concentration of plasma protein and hematocrit; the amount of plasma and blood given in resuscitation reflect to some extent the surgeon's estimate of the gravity of the patient's condition, and the death rate relates in some degree to the severity of shock, though the presence of other etiologic factors tends to obscure this point. In Table XI, these data, as provided by 86 cases, are tabulated and grouped according to the systolic blood pressure readings before treatment. In Table XII, the data are analyzed with plasma volume as the critical factor, while in Table XIII the hematocrit readings are the basis for analysis. From these tabulations, it would appear that systolic blood pressure in untreated wound shock affords the basis of analysis yielding the best agreement between the different factors under consideration.

There are various circumstances which might be expected to raise the nonprotein nitrogen of the blood following grave wounding. Among these are dehydration, sepsis and fever and increased metabolic breakdown, shock and hypotension with decreased renal blood flow, hemorrhage into the gastrointestinal tract, renal damage from sulfonamide therapy and hemoglobinuric nephropathy. As shown in Table XIV, azotemia is often present during the first ten days of convalescence. In some cases this occurs during the first day after wounding, but elevation of nonprotein nitrogen is more pronounced and more common at the end of the first week. In most cases the azotemia is not severe. In Table XXI appear data from a case of severe renal dysfunction which progressed to anuria, with subsequent recovery. Azotemia, hypertension, albuminuria and cylindruria were present and the specific gravity of the urine was low. The patient's condition improved greatly after urine volume became normal, but the subsequent state of his renal function could not be determined owing to his evacuation. Coincident with nitrogen retention in this case, there was sharp elevation of blood sulfonamide content. It is impossible to say what the cause of the renal insufficiency in this case was, but probably it resulted from a combination of unfavorable circumstances. The wounds were extensive and included direct trauma to one kidney, the blood pressure was at zero values on admission to hospital, blood volume was seriously reduced, a large amount (25 Gm.) of sulfonamide was administered in the first 24 hours, and two liters of blood were given before and during operation. From the present study, it is not possible to say how frequently serious renal damage occurs

in the gravely wounded, but in this group of 114 cases there were at least four of them showing some degree of renal impairment.

In the management of these cases, sulfonamide was employed variously, and with uncertain enthusiasm. Theoretically, each wounded soldier received 5 Gm. of crystalline sulfonamide topically and 6 Gm. orally as part of routine forward medical care. At the time of definitive surgical operation from 5 to 10 Gm. of sulfanilamide was applied to wounds of pleural or peritoneal cavity; postoperatively from 2.5 to 5.0 Gm. sodium sulfadiazine was given as a daily intravenous injection for five to seven days, particularly in cases of penetrating abdominal wounds. Actually, this routine was frequently modified in any or all of its parts, and precise knowledge of how much sulfonamide had been used in the course of first-aid and definitive surgical care was never available. The total dosage was a matter of guesswork. For such reasons the data shown in Table XV are of special interest. All of the 89 patients whose blood was examined at various intervals after admission to hospital, supposedly were under sulfonamide therapy. The values for blood concentration indicate that many received little or none of the drug, and the variability of the blood values showed that only dubious and uncertain benefit could be expected.

In several instances the authors were given a chance to note the rapid changes in the blood picture resulting from fulminating clostridial myositis. As edema of the involved and adjacent tissues rapidly develops, plasma protein concentration falls and plasma volume declines, while red cell hematocrit may remain normal, or even rise. The pattern of the blood, thus, may come to resemble that in severe burns. Plasma infusions are clearly an important part of treatment. In Tables XXIII and XXIV, illustrative data are tabulated as provided by two patients with clostridial myositis, clinically diagnosed as due to *C. oedematiens.*

There is considerable evidence at hand showing that the convalescent period immediately after severe wounding is marked by frequent disturbances in fluid balance. Dehydration in varying degree is often present, despite the fact that infusions of 5 per cent glucose solution and physiologic salt solution were part of postoperative care. An examination of the data in Tables XVI to XXIV, inclusive, discloses the frequency with which fluctuations in plasma protein concentration and red cell hematocrit occur. The specific gravity of the urine, as shown in Table XXVI, varied widely but in the main a concentrated urine was excreted during the first four days after wounding. The average specific gravity of the urine in the cases studied was 1.020. The prevalence of dehydration is further indicated by the figures for average daily urine volume and for minimal urine volume during the various days observed, and by the frequency of azotemia and hypochloremia as shown in Tables XVI to XXIV. Since 48 per cent of the group of ground force casualties studied had received penetrating abdominal wounds, disturbances in fluid balance would be expected and in fact did form a challenging problem in surgical after-care.

Included in the plan of study was investigation of the importance of acidosis as a part of the picture of wound shock. Owing to the limitation on available biochemical methods, the question was approached indirectly through analysis of the urine. In a group of 14 patients it was possible to make quantitative collections of urine beginning with the first specimen passed following admission to hospital a few hours after wounding, and to examine individual samples for ammonia content and titratable acid. In addition, a few patients (five) were given 40 Gm. of sodium bicarbonate intravenously during the course of the same studies. From the data given in Table XXVII, one sees that ammonia and titratable acid were present in the urine in quantities sufficient to effect considerable saving of fixed base.[8] The ratio between ammonia and titratable acid is in the normal range, and it is a fair inference that these phases of renal function suffered no serious impairment. The findings are consistent with a presumption that some degree of base-deficiency was the rule in the cases observed. The administration of sodium bicarbonate on the first two days after wounding resulted in a reduction in the amount of ammonia and titratable acid excreted, and adds to the validity of the above concepts.

SUMMARY

Clinical and biochemical investigations were made on 100 ground force casualties and 14 air combat casualties; all were selected cases with severe wounds inflicted by high explosive missiles. The study began immediately after admission to a forward hospital a few hours after wounding and continued for one to two weeks. Data were obtained of significance in the analysis of wound shock, resuscitation, and postoperative care.

CONCLUSIONS

1. The syndrome of traumatic shock due to wounding by high explosive missiles was notable for its variability.

2. Reduction in blood volume was a characteristic finding.

3. Hemoconcentration as evidenced by elevation of red cell hematocrit or plasma protein above normal ranges, was not encountered in the absence of burns or complicating clostridial myositis.

4. Despite restorative therapy, anemia and hypoproteinemia were the rule during convalescence.

5. Quantitative improvements in blood volume and in concentration of plasma protein and hemoglobin did not occur in response to replacement of plasma and blood.

6. No evidence of overdosage in plasma and whole blood therapy was detected.

7. Dehydration and azotemia were common in early convalescence.

8. In fulminating clostridial myositis, with edema, plasma protein concentration and blood volume fell rapidly.

9. Erratic effects were obtained from sulfonamide therapy if the concentration of the drug in the blood is significant.

10. Base deficiency, when present, was of mild degree as evidenced by the urinary reactions.

11. The effectiveness of the surgical management of this selected group of the gravely wounded is attested by the low mortality rate during the period of observation.

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EVACUATION HOSPITAL EXPERIENCES WITH WAR WOUNDS AND INJURIES OF THE CHEST

A PRELIMINARY REPORT

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THE CASES which form the basis of this report were all seen in one Evacuation Hospital over a period extending from November 8, 1942, to August 2, 1944. During this time the hospital was at varying distances from the front lines in the North African and Italian campaigns. In most situations casualties were admitted 2 to 12 hours after injury. At Anzio, when the hospital was only a few miles from the front line and well within artillery range of the enemy, many casualties were received within a few minutes after being wounded. In all, 1,210 patients with wounds and injuries of the chest were treated, this number constituted 6.9 per cent of the total battle casualties and injuries admitted to this hospital. The experience gained in the management of these cases has led to the development of certain policies in regard to preoperative, operative and postoperative care of the patients who have suffered wounds of the chest. It is not our purpose to discuss in detail all the problems of the surgery of thoracic wounds. However, certain fundamental concepts may well be emphasized in this preliminary report. The various types of wounds of the chest will be discussed separately, giving some statistics and, in most instances, illustrating methods of management by case reports.

The rôle we play in a forward installation in respect to chest lesions is to: (1) Save lives. (2) Be conservative. (3) Evacuate patient to Base Hospital when transportable.

GENERALMANAGEMENT OF CHEST CASES

The patient is propped up to a semisitting position if he is not in profound shock or unconscious. All clothing is removed from the chest and abdomen to permit an adequate examination. Immediate attention is given to shock and hemorrhage by starting replacement therapy, preferably with whole blood. The normal relationship of the intrathoracic organs is promptly restored, inasmuch as disturbances here may be responsible for shock. This may be attained, first, by adequately covering open chest wounds, if they exist; and second, by aspiration of blood and air from the pleural cavity. Thoracentesis for blood can be done nicely by using a transfusion vacuum bottle (Fig. 1). Blood so withdrawn is used for autotransfusion; though if an abdominal injury is suspected, the blood is not to be given until thoracotomy has proven the absence of contamination with gastric or intestinal contents. If pressure pneumothorax exists, a trocar-thoracotomy is done, placing the catheter in the 2nd interspace anteriorly in midclavicular line. The catheter is connected to a water-seal bottle.

All chest wounds, or chest wall injuries, with pain, receive intercostal nerve block (4 to 6 cc. of 1 per cent novocaine) two segments above and below the lesion. Paravertebral sympathetic block is occasionally substituted when

the lesion is far posterior. If morphine has not been given previously, ⅙ gr. and atropine sulfate 1/100 gr. are given intravenously.

Excessive secretions and blood in the trachea and bronchi are aspirated by tracheal catheter suction. Occasionally bronchoscopic aspiration is necessary. Oxygen therapy is often useful in combating shock and anoxemia. The measures outlined above will render most patients with wounds of the chest good operative risks. Roentgenograms in two planes are always taken prior to operation. As soon as all indicated measures are carried out, necessary surgery is performed.

Anesthesia of choice in all suspected sucking wounds of the chest is gas-oxygen-ether, with intratracheal tube in position. This is necessary as all penetrating and perforating wounds of the chest are potentially sucking wounds and usually do suck when the débridement of the chest wall is complete. If the positive pressure anesthesia machine is not available, local anesthesia is then used. Open drop-ether and sodium pentothal anesthesia should never be used in the management of these wounds.

Débridement of all chest wall wounds must be thorough, which entails removing all devitalized tissue and loose rib fragemnts. Following débridement sucking wounds are closed by approximating muscle and fascia layers, thus, occluding the defect in the pleura. The skin is also closed when necessary to assure an air-tight closure. It should be emphasized that the pleura itself need not be sutured. Every effort is made to secure and maintain complete expansion of the lung, using positive pressure anesthesia and aspirating any residual air by needle. At the close of operation repeat the intercostal nerve block. Hemothoraces are aspirated without air replacement every 24 to 48 hours until the pleural cavity is free of fluid. Intratracheal catheter is used as indicated in the postoperative period.

PENETRATING AND PERFORATING WOUNDS OF THE CHEST

Penetrating and perforating wounds of the chest, with no indication for emergency thoracotomy, comprise the vast majority of the cases in this series. These cases, for the most part, require only simple débridement of the chest wall wound. The preoperative and postoperative care of these patients is most important.

These cases frequently have an accompanying hemopneumothorax, with associated severe pain (chest wall). Such factors may produce symptoms of shock and definitely aggravate shock when present. Thus, the problem of correcting this pathophysiologic condition should be undertaken immediately and vigorously by: (1) Aspiration of the hemopneumothorax (using blood for autotransfusion). (2) Doing an intercostal nerve block. This allows the patient to breathe deeply and cough painlessly so as to drain the bronchial tree of excessive bronchial secretion that seemingly accompanies chest wall and pulmonary trauma.

The following is an example of a perforating wound of the chest which was treated conservatively:

Case 1.—This patient suffered a perforating wound of the left chest from a 32-caliber bullet. He was admitted to the hospital shortly thereafter in moderate shock, and with some dyspnea. There was no hemoptysis and no gross bleeding. The wound of entrance was one centimeter in diameter at the level of the fourth rib anteriorly in the midclavicular line; the wound of exit was small and just beneath the inferior angle of the right scapula. The wounds were not sucking. No evidence of hemothorax or pneumothorax existed. An intercostal nerve block was done. Attempted aspiration of the chest yielded no blood or air. The patient was readily stabilized without replacement therapy. Under local anesthesia, the wounds were débrided, and no evidence of sucking found. The wound surfaces were powdered with sulfanilamide and vaselined gauze dressings were applied. On the first postoperative day 100 cc. of blood was aspirated from the left chest cavity. On the fifth postoperative day the patient was sitting up, breathing comfortably and had no complaints. He was evacuated to the rear on the sixth postoperative day.

COMMENT: In this case the relief of pain by intercostal nerve block brought an apparently ill patient into a comfortable state. While it is true that blood and fluid do not always produce marked symptoms, time and experience has demonstrated that patients are able to return to duty sooner when the pleural cavity is kept dry and the lung is allowed to reëxpand.

SUCKING WOUNDS OF THE CHEST

Large sucking wounds of the chest demand prompt attention and may present some difficulty in closure. When a wound is large and the chest open, fluid and clots should be aspirated through the wound; furthermore, one should endeavor to be certain that hemorrhage has stopped and that there has been no injury to the diaphragm. Intrapleural foreign bodies and only readily accessible fragments in the lung should be removed; occasionally it is necessary and expedient to enlarge the wound to do this, as exemplified in the following cases:

Case 2.—This patient had a perforating gunshot wound of the left chest, with wound of entrance anterior in the region of the third rib and the wound of exit through the scapula just above the angle. An hemopneumothorax existed and there was also a perforating wound of the left hand. Six hours after being wounded an intercostal nerve block was done, and 400 cc. of blood was aspirated from the left pleural cavity; this blood was autotransfused. (Prior to the nerve block and aspiration atropine sulfate gr. 1/100 and morphine sulfate gr. 1/4 were administered). The blood pressure was 108/70. Ten hours following the wound, operation was performed under endotracheal positive pressure anesthesia. The chest wound was found to be gaping and air and blood were spluttering therefrom. A perforation of the lung lay directly beneath the chest wall perforation and the air was seen to be coming from this wound in the lung. The chest wall wound was enlarged and the three-inch laceration of the lung, containing a bronchial fistula, was closed with interrupted silk sutures. Closure of the chest wall wound was done in anatomic layers with catgut, and the skin was closed with silk sutures. A small catheter was placed in the third interspace in the midclavicular line. The posterior wound was then débrided and also the wound of the right hand. At the end of the procedure the blood pressure was 104/78. The patient was given penicillin 25,000 units intramuscularly every three hours. On the second postoperative day good breath sounds were heard throughout the posterior chest. The anterior catheter which had been connected with a water-seal bottle immediately postoperatively had ceased to drain four days postoperatively; it was removed at that time. On the fifth postoperative

day the patient had a temperature of 103° F., and signs of fluid posteriorly, 1,000 cc. of thin bloody fluid was aspirated from the left chest. The patient was evacuated in good condition, with the note that further thoracentesis might be necessary.

COMMENT: Too much dependence was placed on the anterior catheter to drain the chest cavity. The patient's position had been changed to effect fluid drainage, but without success. If daily thoracentesis had been done he should have been relatively dry at the time of evacuation.

Case 3.—This soldier was wounded by a machine gun bullet, and was admitted to the hospital in severe shock, with a blood pressure of 40/0 and shallow respirations (30 per minute). The wound of entrance was in the left chest at the lateral border of the left scapula at the level of the fourth rib. The foreign body could be palpated in an hematoma the size of a golf ball in the right chest wall in the posterior axillary line at the level of the fourth rib. Three and one-half hours after being wounded (0800 hours) an intercostal nerve block was done. At 1245 hours atropine sulfate gr. 1/100 was administered intravenously and 1,300 cc. of blood was aspirated from the left pleural cavity and given as autotransfusion in addition to 600 cc. of whole blood and 250 cc. of plasma. The blood pressure then became stable at 100/60. The patient vomited and coughed up a blood-tinged milk-like substance. The presence of chyle from a thoracic duct wound or sulfathiazole from an esophageal wound was considered. The patient was given a small amount of barium for roentgenologic examination, but no lesion in the esophagus could be demonstrated.

At 1330 hours of the same day, under oxygen-ether intratracheal anesthesia, the wound was débrided. It was found to extend through the scapula and it was necessary to reflect the scapula through a regular thoracoplasty incision to complete the débridement. The lower border of the fourth rib and the fourth intercostal bundle were found to be destroyed and a sucking wound 4 cm. in length was found. Two hundred cubic centimeters of blood were aspirated from the left pleural cavity. The patient's condition did not warrant further exploration of the chest cavity. The sucking wound was closed by approximation of muscle and fascia over it. The machine gun bullet was then removed from the right chest wall. Six hundred cubic centimeters of additional blood was given during operation. The patient was returned to ward in good condition. Twenty-five thousand units of penicillin was administered intramuscularly every three hours. He was evacuated to the rear on the third postoperative day, with a dry chest.

Case 4.—This patient was wounded by artillery fire. Dyspnea, weakness and pain in the abdomen developed immediately. He was admitted to the hospital one hour and forty-five minutes after being wounded, and was in fairly good condition. The abdomen was spastic, but there was no rebound tenderness. There was a penetrating wound 2 cm. in diameter in the left posterior scapular line at the level of the ninth interspace. There were two small perforating wounds in the left arm. There were signs of a large hemothorax, but no blood could be obtained by aspiration. The intercostal nerves in the region of the wound were blocked. Then, under oxygen-ether anesthesia, with intratracheal tube in place, the wound of the left posterior chest wall was excised. There was much destruction of the muscle and the tenth rib. With débridement of the wound a large clotted hemothorax was revealed. Approximately 1,800 cc. of clotted blood were evacuated from the left pleural cavity. A puncture-like laceration was found in the lingula of the left upper lobe. This laceration was 5 cm. in diameter, and was bleeding freely and bubbling air. The lacerated lung was repaired with interrupted silk sutures. The diaphragm was not involved. A catheter was placed in the eighth interspace in the posterior axillary line. Sixty thousand units of penicillin was introduced intrapleurally. The incision was closed in anatomic layers with catgut sutures. Six hundred cubic centimeters of blood and 250 cc. of plasma were given during operation. The patient returned to ward in good condition. Penicillin therapy was

continued by the intramuscular route, and the catheter was removed 48 hours postoperatively. There were some signs of fluid at the base, but only 20 cc. of thin fluid could be aspirated. Breath sounds were present anteriorly and posteriorly. The patient was evacuated to the Base on the sixth postoperative day.

COMMENT: The size of the wound after adequate débridement of the chest-wall permitted evacuation of a large clotted hemothorax, repair of a laceration of the lung and a bronchial fistula, and inspection of the diaphragm.

The above cases represent most of the common problems encountered in the sucking chest wound and illustrate the usual method of handling them. When there is extensive destruction of muscle and ribs, proper débridement of the wounds ordinarily opens the pleural cavity; then an estimation of the intrathoracic damage is made. Frequently, any necessary repair can be effected by enlarging the wound slightly.

THORACOTOMY INDICATIONS AND APPROACH

There are not many indications for formal emergency thoracotomies in Forward Hospitals. In fact, we believe there are only four, namely: (1) Cases with continued gross bleeding. (2) Uncontrolled bronchopleural fistulae (such as lacerated bronchi). (3) Suspected and known thoraco-abdominal lesions (which will be discussed separately). (4) Suspected and known esophageal wounds.

Under sucking wounds certain indications were mentioned for enlarging those wounds and such enlargements are properly thoracotomies, but we ordinarily think of them as extensive débridements; of these and formal thoracotomies there were 110 cases (which constituted 10 per cent of the group), not including the thoracoceliotomies.

The elective removal of foreign bodies and decortication of organized hemothorax are not performed routinely in an Evacuation Hospital, but in situations where Base Hospitals have been far removed, it has been expedient to retain these patients for definitive surgery.

When thoracotomies are indicated, in some instances they may be done through the wound of the chest wall, provided the lesion is in the site of the elective incision (*i.e.*, posterolateral area). This requires enlargement of the débrided wound. Against this procedure is the fact that these wounds frequently break down from infection. For this reason, when a large thoracotomy incision must be made, it should, whenever possible, be kept away from the original wound. Indications for thoracotomy will be demonstrated by illustrative cases.

HEMORRHAGE FROM LARGE INTRATHORACIC BLOOD VESSEL

When there is evidence of bleeding from a large intrathoracic blood vessel (this largely determined by route taken by missile—usually through the neck and supraclavicular region—and by the fact the hemothorax is clotted), the case should be explored as soon as the blood pressure becomes stabilized. Procrastination in such cases, in our experience, has been disastrous, as illustrated in the following two cases:

Case 5.—This patient was wounded by shell fragments. He received a penetrating wound of the left chest with the entrance wound in the left neck, and also penetrating wounds of the right upper arm and the left hip. He was admitted to the hospital with a large hemothorax five hours after having been wounded. After several unsuccessful attempts to aspirate this hemothorax it was decided the blood was clotted. The wound of the neck was débrided, and the tract partially explored where it ran superior to the first rib. The wound was powdered with sulfanilamide and covered with a vaselined gauze dressing. The first three days postoperatively no aspiration of the chest was attempted; 400 to 500 cc. of blood were aspirated daily from the left chest for the next five days. The patient was supported by numerous blood transfusions. On the eleventh postoperative day 900 cc. of thin bloody fluid were aspirated. On the twelfth postoperative day the patient suddenly went into severe shock; the radial pulse was imperceptible and no blood pressure reading could be obtained. Respiration was slightly labored, and the skin was cold and clammy. The breath sounds on the left were distant. The patient was given 1,000 cc. of blood, and he rallied satisfactorily. Attempted aspiration was unsuccessful. The following day his pulse was 148. Four hundred cubic centimeters of blood-tinged fluid was aspirated. Under oxygen-ether-intratracheal anesthesia a sickle-shaped incision was made from the middle third of the clavicle to the fifth costosternal junction. The first rib was excised at the sternocartilaginous junction. As the pleural cavity was entered copious quantities of blood poured out. Rapidly, the second, third and fourth ribs were severed at the sternocartilaginous junction. A finger was used to compress a rent in a bleeding vessel at the apex of the chest. The bleeding was from the region of the subclavian artery and innominate vein. Apparently, local infection had completed the laceration of these vessels. The bleeding was controlled eventually by gross clamping and mass sutures at the apex of the chest. The chest wall closure was effected with catgut sutures for muscle and fascia and silk sutures for the skin. A catheter was placed in the eighth intercostal space. The patient's blood pressure was imperceptible through much of the operation, although intravenous blood was started prior to operation in both ankles and the right arm; 3,000 cc. of blood were given during operation and 750 cc. immediately following operation. One hour postoperatively the blood pressure was 100/60, and four hours postoperatively it was 102/80. Oxygen was discontinued one hour after the operation.

The postoperative course was uneventful the first 13 days. When the hospital blew down in a snow storm this patient, with 700 others, was evacuated to another hospital. The catheter was not draining at this time. He was transferred to a General Hospital four days later. On admission there the following note was made: "Dyspneic, distressed, with intercostal catheter not draining. On the twenty-first postoperative day a large tube was inserted in the eighth rib bed; the pus which drained was putrid. There was no bronchial fistula and the anterior wound was nicely healed. Steady improvement followed this procedure."

COMMENT: The direction taken by the missile and the early clotting of blood indicated the possibility of a large vessel laceration. However, in the absence of shock and other evidence of gross bleeding, thoracotomy seemed contraindicated at the time. Subsequent events suggest that it would have been the better procedure. Intravenous fluids started in three veins before surgery unquestionably saved this patient's life.

Case 6.—This patient was admitted to the hospital two hours after sustaining shell fragment wounds; one of which was a 3- x 2-cm. penetrating wound in midline of the neck above the thyroid cartilage and the other a 1- x 2-cm. penetrating wound in the right lateral portion of the neck in the same plane. On admission, he was not in shock or experiencing any respiratory distress. Attempted aspiration was unproductive. Five hours after having been wounded, under intratracheal oxygen-ether anesthesia, a thy-

roidectomy-type incision was made and the right sternocleidomastoid muscle divided. Diffuse bleeding, which was thought to be coming from the right innominate vein, was in evidence beneath the right clavicle. Bleeding was controlled by a gauze pack, which was left in place. At completion of this débridement blood pressure was 92/68. Eighty cubic centimeters of blood was aspirated from the right chest; however, more blood remained, which was clotted and unaspirable. One hour postoperatively the patient went into profound shock and he expired ten minutes later.

Autopsy revealed intrathoracic laceration of the right subclavian artery and innominate vein; the right thoracic cavity was filled with clotted blood. Massive hemorrhage was the immediate cause of death.

COMMENT: This patient should have had a formal thoracotomy, and an attempt made to ligate the lacerated vessels. In cases such as these, it is advisable to start blood and plasma in every available arm and leg prior to operation.

LACERATED BRONCHI WITH BRONCHOPLEURAL FISTULA

Any case with pressure pneumothorax which cannot be controlled by needle aspiration or by a large catheter in the anterior chest should have an immediate thoracotomy.

We think it is noteworthy to report these two cases of lacerations of main stem bronchi, with large defects, because it is only in rare instances that such patients live to be evacuated from the battle field. At the Anzio Beachhead, hospitals occupied a peculiar position, in that they were part of the battle field, and patients were injured on or adjoining the hospital area. These are the only cases of main-stem bronchi defects, to our knowledge, to reach a hospital alive. Most certainly, such cases deserve emergency thoracotomies.

Case 7.—The patient was received at this hospital one hour after sustaining a shell fragment wound of the right chest, eighth interspace, posteriorly. Presumably, he was in a prone position when wounded. He immediately became aware of a sucking wound of the chest and inability to move his lower extremities. Dyspnea followed. On admission, he was in shock and suffering profound respiratory embarrassment. Complete paralysis below the eighth dorsal segment existed. A large 5- to 6-cm. diameter wound in the eighth interspace posteriorly was sucking air, draining blood and was filled with clothes and bone fragments. The sucking was stopped with a vaselined gauze pack to the wound. A paravertebral nerve block of the sixth to tenth intercostal nerves improved the respirations and the patient began to cough up a large amount of blood. Twenty-five grams of serum albumin elevated the blood pressure to 96/52, and the chest was immediately aspirated of 800 cc. of blood, which was given as an autotransfusion. Insertion of an intercostal tube relieved the developing pressure hemothorax. The patient became fairly well stabilized nine hours after injury, when suddenly pneumothorax increased, and operation then became an emergency. Under intratracheal oxygen-ether anesthesia the wound in the region of the eighth rib was excised and a posterolateral thoracotomy incision in the right eighth interspace was made. A large ragged bullet-shaped shell fragment, 12 cm. in length, was found penetrating a main-stem bronchus from which air and blood was flowing freely. The azygos vein was found to be lacerated and was sutured, but not completely occluded. There was an irregular 2.5-cm. defect in the posterolateral wall of the right main-stem bronchus just distal to the bifurcation of the trachea. This defect was approximated, after considerable difficulty, with interrupted silk sutures and reinforced with muscle. The repair was water-tested and no leakage experienced. A 12- to 14-cm. long transverse laceration of

the upper lobe was closed with interrupted silk sutures. The lung was then reexpanded, except for a portion of the upper lobe in the region of the lacerated area. The closure of the incision was done in anatomic layers with catgut sutures for deep layers and silk sutures for skin. The patient was in fair condition at termination of the operation. He had received 1,000 cc. of blood and 250 cc. of plasma during the procedure.

The patient underwent a stormy postoperative course, with abdominal distention and the accompanying sequelae of a cord transection. Seven days postoperatively a suprapubic cystostomy was performed and a hip spica applied to insure the patient's

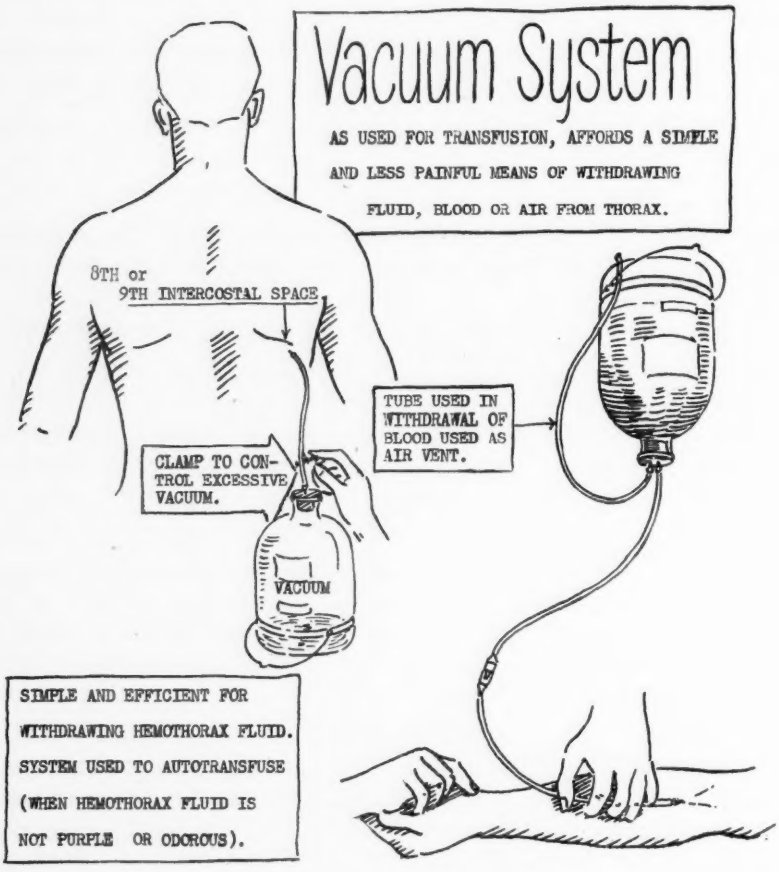

FIG. 1

comfort and to facilitate his handling in evacuation to the rear. Although the right lung remained expanded, on the twelfth postoperative day an infection of the incision developed at the site which included the original wound (experiences such as this illustrate the inadvisability of including the wounded area in the thoracotomy incision). An empyema ensued. Four days later he was sufficiently stabilized for evacuation to the rear. It should be noted that he had received 25,000 units of penicillin intramuscularly every three hours for ten days postoperatively. No sulfonamides were given (when penicillin is available, sulfonamides are never used). Reports a month later stated there was an empyema cavity of 200 to 300 cc. remaining, and there was some return of sensation in the extremities. A progress note four months later recorded progress as "excellent."

Case 8.—This soldier was admitted to a hospital one hour and 50 minutes after receiving multiple penetrating shell fragment wounds, most notable of which was a large sucking wound of the seventh interspace posteriorly that involved the seventh Dorsal vertebra. Complete paralysis existed from the eighth dorsal segment downwards. Being in moderate shock, he was sustained with serum albumin and transfusion until his blood pressure became stable. He was received at this hospital in a dyspneic state, with intercostal catheter working and with air and blood in the right thorax. His chest was very "wet' and copious quantities of bloody exudate were aspirated. Then, under intratracheal oxygen-ether anesthesia, two hours after admission here, the right pleural space was opened at a selected site in the fifth intercostal space; 700 cc. of blood were evacuated from the pleural cavity. Air was seen bubbling from the right main-stem bronchus. There was a 2-cm. defect in the anterior and left lateral wall where a piece of the bronchus had been torn out approximately 1.5 cm. from the bifurcation of the trachea. With considerable difficulty, three heavy silk sutures were placed to occlude the defect (water-tested) and a muscle graft was sutured over the area. There was profuse bleeding in the wound exit in the seventh intercostal space at the level of the transverse process. This wound extended to the spinal canal and the cord could be visualized, but no damage was apparent. Bleeding was controlled only by a pack which was brought out through the wound. The pleura was then closed and the ribs approximated. Sixty thousand units of penicillin were deposited intrathoracically. Two catheters were inserted; one in the eighth interspace posteriorly, the other in the second interspace in the midclavicular line anteriorly. The patient had received 1,200 cc. of blood and 1,000 cc. of glucose during operation. Penicillin therapy, 25,000 units intramuscularly every three hours, was instituted. Forty-eight hours postoperatively the patient became irrational, had a temperature elevation to 104° F., and developed a stiff neck with other signs and symptoms of meningitis. Seventy-two hours postoperatively the temperature remained elevated to 105° F., stiffness of neck persisted and the general condition became more critical. At this time the hospital was forced to move forward; the patient was left at a Holding Hospital, and placed on the "seriously ill" list. His lung remained completely aerated. The patient had been receiving 25,000 units of penicillin intramuscularly every three hours throughout his hospital stay.

POSTOPERATIVE CARE

After operation, intercostal nerve block should be repeated and residual air aspirated by suction bottle in the 2nd intercostal space in the midclavicular line. All thoracotomies are drained with a catheter in the 7th or 8th intercostal space in the posterior axillary line. Postoperatively, all patients are aspirated with a long catheter which is inserted through the endotracheal tube or are aspirated by means of a bronchoscope. It goes without saying that bronchoscopic aspiration is more complete although it entails prolonged anesthesia and consumes more time, which is a real factor when there is a large back-log of untreated wounded. In our experience the long catheter aspiration has proven satisfactory.

THORACO-ABDOMINAL WOUNDS

There were 103 cases with coexisting wounds of the chest and the abdominal cavity (two died prior to operation). In six of these cases the wounds were caused by two or more missiles without injury to the diaphragm. The injuries below the diaphragm were limited largely to the upper abdominal viscera. In the treatment of these cases attention was focused primarily on

the chest; measures to relieve respiratory difficulties were instituted promptly. These consisted chiefly of insuring that the dressing adequately occluded the chest wound, aspirating the hemopneumothoraces, autotransfusion, and novocaine block of intercostal nerves in areas of the chest wounds. Relief of respiratory difficulty and treatment of shock were carried on simultaneouly. With improvement in the patient's general condition an appraisal of the chest and abdominal injuries was made. The choice of approach to abdominal injury was determined largely by the fancy or experience of the surgeon. From our experiences with this group of cases, after studying in detail their postoperative course and reviewing the mortality figures, the claims of those who strongly advocate one approach over the other cannot be dogmatically substantiated, though for those familiar with the chest, the thoracic approach is definitely preferable. Forty thoraco-abdominal wounds were treated by thoracoceliotomy (all work done through chest incision), with ten deaths (25 per cent). Fifty-four thoraco-abdominal wounds were treated primarily through the celiotomy incision, with 17 deaths (31 per cent). Six thoraco-abdominal wounds were treated through a combined approach, with two deaths (33 per cent). Major Pat R. Imes, in preparing a report on the abdominal wounds treated in this hospital, has noted the high mortality that accompanies multiple abdominal visceral wounds. His figures indicate that mortality is in direct proportion to the number of viscera involved. The superior statistics for the thoracic approach are seen to be more apparent than real when the following table is studied:

	Wound of:								
	Abdomen Negative			One Abdominal Viscus			Two or More Abdominal Viscera		
	Cases	Deaths	%	Cases	Deaths	%	Cases	Deaths	%
Celiotomy	4	0	0	26	3	11	24	14	58
Left thoracoceliotomy	4	0	0	15	4	27	8	4	50
Right thoracoceliotomy	0	0	0	10	0	0	3	2	66
Total	8	0	0	51	7	14	35	20	57

One flank wound lacerated the diaphragm. Nephrectomy and repair of the diaphragm were accomplished through a kidney incision.

These mortality figures indicate a somewhat higher death rate than those from abdominal wounds without involvement of the chest. But on comparison with similar upper abdominal visceral injuries there is no remarkable difference. However, the fact remains that the chest wound must first be converted into a nonsucking wound and the physiology of the chest stabilized prior to a thoracoceliotomy or abdominalceliotomy.

A—THORACIC APPROACH, LEFT-SIDED

No. of Cases: 27 *Deaths: 8*

Thoracic approach on a left-sided lesion is quite adequate unless the missile is directed from below the umbilicus upwards; a rare condition.

Case 9.—This soldier was admitted to the hospital with a severe perforating gunshot wound of the chest, with lacerations of the diaphragm, spleen, left lower lobe of lung and herniation into the chest of the stomach, spleen, colon and small bowel. Three hours after being wounded, under endotracheal ether anesthesia, a left-sided thoracotomy was performed with resection of eight inches of the ninth rib. The chest cavity was found to contain the entire stomach, tremendously dilated, the omentum and transverse colon with splenic flexure and a portion of the jejunum. The badly lacerated spleen was removed, and a stomach tube reduced the stomach 60 per cent in size. The left gastro-epiploic artery which was torn near its termination was ligated. The kidney, colon, stomach and bowel were found to be intact. The torn lesser omentum was repaired, and the diaphragm was closed in two layers with silk sutures. The tip of the lower lobe of the left lung, which was badly torn and bleeding, was resected and sutured with swaged catgut. A catheter was inserted intercostally, and closure was effected with paracostal sutures and suture of the muscle layers. Twelve days postoperatively the patient was evacuated to a Base Hospital.

COMMENT: This case rather typifies left thoraco-abdominal lesions, and illustrates the proximity of all left upper quadrant organs to the chest cavity and their accessibility through this approach. When the diaphragm is opened these organs actually ooze into the chest cavity.

Case 10.—In this instance a soldier received a shell fragment wound which penetrated the tenth interspace in the posterior axillary line. At a Forward Field Hospital exploration of the wound was done and a perforation of the diaphragm sutured. The wound was then hurriedly closed, apparently because the hospital was under shell fire. On admission to this hospital, 24 hours after being wounded, the patient was suffering severe abdominal pain, with distention. Accompanying the patient was a note by the surgeon stating that "he thought a celiotomy should be performed." Through a left rectus incision the abdomen was opened, and the peritoneal cavity found to contain clean blood although all viscera appeared intact. Thirty-six hours after operation the thoracotomy wound became edematous; crepitation and a foul odor were present. Upon exploration of the wound, the superficial muscle was found to be necrotic and characteristic gas gangrene was seen to exist. The patient expired 48 hours postoperatively. Findings at the autopsy revealed gas gangrene of the chest wall, and a perforation of the splenic flexure of the colon.

COMMENT: This case represents an injury to the retroperitoneal portion of the splenic flexure that was not identified through a celiotomy. This could have been handled simply, and easily, through a thoracotomy approach. That the lesion was not recognized is not the fault of the thoracic approach.

B—THORACIC APPROACH, RIGHT-SIDED

No. of Cases: 13 *Deaths: 2*

Thoracic approach for a right-sided thoraco-abdominal lesion is usually the procedure of choice.

Case 11.—After having sustained a penetrating wound of the right lower chest region the patient was admitted to the hospital shortly thereafter, with a rigid abdomen. The tenth right rib was resected and a laceration of the lower lobe of the lung sutured. The diaphragm was found to be perforated and the foreign body to lie in a tear in the liver. Inspection of the hepatic flexure, duodenum and kidney disclosed no injury, nor were other intra-abdominal viscera injured. The liver was drained subdiaphragmatically and the diaphragm closed in two layers with interrupted silk sutures. The patient was evacuated to the rear six days later in good condition.

COMMENT: In the 17 cases where right thoraco-abdominal lesions existed it was necessary to make an additional abdominal incision in only five instances.

C—ABDOMINAL APPROACH

No. of Cases: 54 *Deaths: 17*

Case 12.—This patient was admitted to the hospital, with a blood pressure of 90/50 and pulse of 140, after having experienced a gunshot wound of the left chest and abdomen. Prior to admission here he had received 500 cc. of plasma. Upon arrival at this installation no respiratory difficulty was apparent and breath sounds were present, though diminished on the left. The abdomen appeared somewhat spastic and moderately tender. The patient complained of suprapubic pain. A perforating wound of the left chest existed with a wound of entry 1.5 cm. in diameter, in the seventh interspace anteriorly, two inches from the midline, and a ragged wound of exit, 7.5 cm. in diameter, in the ninth interspace, just medial to the posterior axillary line. Twenty-five to 30 cc. of blood and a small amount of air were aspirated from the left pleural cavity; 500 cc. of plasma administered and a blood transfusion was begun prior to operation. Then, under ether-oxygen anesthesia, a subcostal incision was made. The peritoneal cavity was found to contain approximately 800 cc. of blood, which was mixed with gastric contents. Exploration disclosed lacerations of the spleen, diaphragm and stomach, which was distended. Entry and exit wounds in the cardia of the stomach were closed and inverted in two layers with chromic catgut. The lesser peritoneal cavity was not involved. The spleen was then delivered and a 6-cm. sucking laceration of the posterior portion of the diaphragm presented itself. The spleen was removed. A large laceration of the diaphragm was closed with difficulty, while another nonsucking laceration of the anterior diaphragm was easily repaired. The closure of the abdomen was done in layers. The patient's condition was not satisfactory during the entire operative procedure, and he expired on the operating table while the skin sutures were being placed. The operation lasted 90 minutes, the greater part of which was consumed in effecting closure of the diaphragm. At no time did the patient's blood pressure exceed 90/60, and in spite of constant administration of blood there was a gradual fall in the blood pressure during the last 30 minutes of the procedure.

COMMENT: All patients with suspected perforations of the diaphragm should have an intratracheal tube in place. The sudden collapse of the left lung in an already shocked patient was directly conducive to the above fatality. The large sucking diaphragmatic perforation was not apparent until the lacerated spleen was delivered for removal. Upon the delivery of the spleen the tamponade of this organ to the perforation of the diaphragm was thus destroyed, and immediately a large sucking wound presented itself in an inaccessible position. The diaphragmatic laceration had to be more or less ignored, except for an ineffective gauze pack, until after the spleen was removed. Two things are apparent: This patient should have had an intratracheal tube, with facilities for maintaining positive intrapulmonic pressure present during operation. This case could have been handled very easily through a thoracic approach.

Case 13.—This soldier was admitted to the hospital nine hours after being wounded by shell fire. Examination disclosed a 1-cm. penetrating wound of the chest in the eighth interspace in the anterior axillary line, with a fragment lying posteriorly at the level of the first lumbar vertebra; also, a perforating wound of the left thigh, a penetrating wound of the right thigh, and a penetrating wound of the left testicle. On

arrival here the patient's blood pressure was 92/30. One thousand cubic centimeters of blood and atropine sulfate gr. 1/100 were administered intravenously. Chest aspiration produced 150 cc. of blood, but no air. (As chest pain was not a factor in this case, a nerve block was not indicated.) Nine hours after the wound was incurred, under intratracheal oxygen-ether anesthesia, the wounds of the chest were débrided and closed; a left subcostal incision was made; and the lacerated and bleeding spleen was removed. The kidney was also found to be torn. (Microscopic examination of the urine showed 8 to 12 red blood cells per high powered field.) A drain was inserted through the flank retroperitoneally. Examination of the stomach, colon and small intestine disclosed no injury. The perforation in the anterior portion of the diaphragm was repaired with catgut sutures and the abdominal incision closed with catgut and silk retention sutures. The wound of the left testicle was débrided and the left testicle removed. The perforating wound had entered the right compartment of the scrotum, but the right testicle was only slightly injured. A rubber drain was inserted through the lower portion of the scrotum. The wounds of the thighs were débrided and foreign bodies removed. Immediately following operation 1,000 cc. of air were aspirated from the left chest cavity. The patient had received 1,000 cc. of blood during operation. Postoperatively, he was placed on penicillin therapy, 25,000 units intramuscularly every three hours. On the second postoperative day an aspiration of the chest cavity yielded 200 cc. of bloody fluid. On the eleventh postoperative day the patient was evacuated to the rear in good condition.

COMMENT: This case was properly managed, as the chest wound was closed prior to celiotomy and the chest aspirated. These abdominal injuries could have been managed easily through a thoracic incision.

CHEST WALL WOUNDS

Chest wall wounds without intrapleural penetration and without contusion of the lung are handled as any other minor wounds. They do not constitute a large group of cases. The wound tract is excised, foreign bodies removed and the wound left open. One should not overlook intrathoracic disturbance merely because the superficial wound appears benign. There are some chest wall wounds without pleural penetration in which there is a varying degree of contusion or local blast injury to the underlying lung. These wounds may be quite serious and demand the same attention and care as intrathoracic wounds.

Case 14.—This patient stated that he was picked up by Aid men one to two hours after being struck by shell fragments in the right shoulder and the left knee. He arrived at the hospital 11 hours after sustaining his wounds, with a moderate hemothorax and "wet" lungs apparent. Many loud râles and rhonchi were heard. There existed a perforating wound of the right upper chest. Roentgenologic examination disclosed comminuted fractures of the posterior portion of the second, third and probably fourth ribs, with mild hemothorax on the right side; also an epiphyseal fracture of the left tibial tubercle. No foreign bodies were visible on either of the films. Fourteen hours after being wounded, under 1 per cent novocaine anesthesia, the 3-cm. wound of entrance just below the outer third of the right clavicle was excised. The tract entered and was traced below the pectoralis muscle, which was laid open. A small fractured piece of the clavicle was removed. The path of the fragment traveled posteriorly, anterior and superior to the axillary vessels and nerves and made a 3-cm. exit wound just medial to the posterior axillary line. The wound was widely excised and a large amount of clotted and liquid blood was evacuated. The tract led through the latissimus dorsi and

trapezius muscles but did not enter the chest cavity. Hemostasis was secured. Sulfanilamide was powdered over both the wounds and a vaselined gauze dressing was applied to the anterior wound. A paravertebral injection of 5 cc. of 1 per cent novocaine was done at levels of the cervical 7 thoracic 1, 2, 3 and 4 vertebrae. Five hundred cubic centimeters of blood were aspirated through the seventh intercostal space in the posterior axillary line and given as an autotransfusion, in addition to 500 cc. of normal saline. The wound of the left knee was then débrided. The patient's bronchial tree, which was filled with secretions, was sucked dry through a bronchoscope; he was returned to the ward in good condition. Four days postoperatively paravertebral block of the right dorsal 2, 3, 4 and 5 was done for relief of pain. No fluid was obtained on attempted aspiration. On the eighth postoperative day aspiration of the right thorax yielded 1,350 cc. of bloody fluid, and two days later 150 cc. of thin pink fluid was withdrawn. On the twelfth postoperative day aspiration of the right chest produced 725 cc. of thin pink fluid; thereafter the chest remained dry. On the twenty-second postoperative day the patient was ambulatory, but still had some loss of function of his right upper arm. He was evacuated to the rear with his chest in satisfactory condition.

FRACTURED RIBS

Of the large number of cases with simple and comminuted fractures of the ribs there were nine who had paradoxical breathing. Pain was controlled with intercostal nerve block. (In view of the fact that simple intercostal nerve block is so effective, we think it is most inadvisable to use adhesive tape to control pain in any chest injury.)

Case 15.—This patient was received at this hospital one hour after suffering injuries from a motorcycle accident which rendered him unconscious. Upon admission here he was dyspneic, cyanotic and experiencing extreme pain with paradoxical breathing. No chest perforation or sucking wound was apparent. There were fractures of the seventh, eighth, ninth, tenth and eleventh ribs, with hemothorax. One per cent novocaine injection of the seventh to eleventh intercostal nerves immediately relieved the pain, and stopped the paradoxical respirations. The patient was autotransfused with 500 cc. of blood on the first day following injury, 600 cc. on the fourth day and 250 cc. on the eleventh day, following which he was free of pain and evacuated to a convalescent hospital.

COMMENT: This case represents a simple, but gratifying means of treating a common injury, even in civilian life.

"BLAST LUNG" INJURIES

"Blast lung" injuries have been a relatively frequent finding, and a very discouraging condition. These cases typically present the following general points: (a) Proximity to a violent explosion such as a land mine; (b) multiple fractures; and (c) delayed respiratory-circulatory collapse, with marked pulmonary edema occurring two to three days after injury. No known therapy has been successfully employed in the treatment of these cases. Positive pressure oxygen therapy has been used with equivocal benefit. A case report, with pathologic findings is recorded below:

Case 16.—This patient was riding in the back seat of a command car when the rear wheel ran over a land mine. He recovered consciousness in a few minutes. He did not experience any dizziness, headache or blurring of vision, but did have difficulty in hearing in both ears. The latter might have been the result of a head injury which

caused a small puncture wound anterior to the left ear near the temporal artery. Speech and cerebration were not impaired. He was treated at a Collecting Station, and both legs and ankles were supported by wire splints extending above the knees. Under ether anesthesia, at a Field Hospital the wound of the right leg was débrided and plaster encasements were applied to both legs (the fracture of the left leg was not compound).

On admission to this hospital, 52 hours after injury, the patient was in good condition, with no complaints. The encasements on both legs were satisfactory, and the toes of both feet were warm. Four hours after admission the patient was conversing normally and rationally. His pupils were equal and regular. His pulse varied between 125 and 130. The following morning the pulse was 140, respirations 32, and blood pressure 98/50. Five hundred cubic centimeters of plasma were administered, after which the pulse was 120, respirations 40, and blood pressure 98/50. The patient had a temperature of 100.4° F., and was very drowsy, but mentally clear when aroused. There was no evidence of a head injury, except a small puncture wound above the left ear. Seven hundred and fifty cubic centimeters of plasma, 500 cc. of blood, and elevation of the foot of his bed brought the blood pressure up to 110 systolic. One and one-half hours later the blood pressure fell to 70 systolic. His respiratory distress was only moderate, although breath sounds were "spottedly" distant and coarse râles were heard throughout the entire chest. The patient was voiding involuntarily and spitting frothy blood.

The upper abdomen was distended, and no bruises or evidence of trauma to the abdominal wall were apparent. There was no peristalsis. Questionable consolidation of both bases with marked friction rub existed. A catheterized specimen of urine was clear. The same evening, at 1900 hours, the patient's condition became critical, with respiratory distress rapidly increasing. Two ampules of coramine and 500 cc. of blood were administered. Diminished breath sounds and tactile fremitus of the left chest with a loud to-and-fro friction rub was felt and heard. Dyspnea and slight cyanosis continued. Oxygen therapy was instituted. The blood pressure was 90/40, pulse 136, and respirations 26. There was a question of a pulmonary infarct. He expectorated frothy sputum with bright blood streaks, and breathed with difficulty and duress. He was lucid enough to attempt to answer questions. The abdomen was moderately distended. The patient struggled for breath (not obstructive type of dyspnea), then suddenly stopped breathing. Artificial respiration failed to revive him. He expired three days after sustaining injuries.

Autopsy revealed a clean peritoneum with no evidence of injury to the retroperitoneal tissues. The examination of the chest disclosed both pleural cavities intact, lungs expanded, grossly hemorrhagic, but crepitant in areas. There was some frothy serous fluid in the bronchial tubes on sectioning. There was no free blood in the pleural cavities. The pulmonary arteries contained thrombi, apparently postmortem. No injury to the thoracic cage was evident, nor was the diaphragm injured. The heart was grossly normal except for a questionable thrombosis in the auricle.

Pathologic Diagnoses.—Gross: (1) Compound fracture, right ankle. (2) Simple fracture, left ankle. (3) Blast injury, lungs (possible). (4) Thrombosis, pulmonary arteries (?). (5) Thrombosis, cardiac auricle (?).

Histiologic Examination.—The lung shows extensive areas of intra-alveolar extravasations of red cells intermingled with varying numbers of pigmented macrophages. Occasional alveoli are filled with fluid containing a few red cells. The bronchial lumina contain large numbers of red cells. Interspersed with blood filled alveoli are occasional emphysematous alveoli, some with ruptured walls.

Pathologic Diagnoses.—Microscopic: (1) Pulmonary hemorrhage, severe, with ruptured alveoli. (2) Congestion of spleen. (3) Epicardial hemorrhage of the heart.

COMMENT: Some so-called "blast lungs" have had fat embolism. Characteristically, these patients also have had compound fractures, usually resulting from land mine traumatic amputations. Unfortunately, the special

fat stain was not used on this tissue. In view of the fact that there were ruptured alveoli we are obliged to classify this as a blast injury of the lungs.

SUMMARY

A preliminary report is made on the management of 1,210 cases of wounds and injuries of the chest. Illustrative case records are presented. The following points are emphasized:

A. Preoperative Recommendations:
   1. Large occlusive vaselined gauze dressing over wound.
   2. Whole blood for shock.
   3. Immediate aspiration of hemothoraces (without air replacement).
   4. Autotransfusion with thoracentesis blood.
   5. Trocar-thoracotomy for pressure pneumothorax.
   6. Intercostal nerve block.
   7. Tracheal and bronchial aspiration.
   8. Oxygen therapy.

B. Operative Recommendations:
   1. Endotracheal anesthesia.
   2. Intravenous fluids, preferably blood, flowing in one or two veins, during operation.
   3. Permissible to enlarge débrided sucking wound for exploration of diaphragm or the control of hemorrhage.
   4. Conservative surgery, minimizing the importance of foreign body removal, unless it is large, accessible, or in a vital organ.
   5. Formal thoracotomy: (1) To control hemorrhage. (2) To close large, bronchopleural fistula. (3) In known or suspected thoracoabdominal lesions. (4) In known or suspected esophageal lesions.

C. Postoperative Recommendations:
   1. Bronchial aspiration (catheter or bronchoscopic).
   2. Aspirate all air immediately postoperatively.
   3. Repeat intercostal nerve block.
   4. Continue nasal oxygen if dyspneic.
   5. Penicillin 25,000 units every three hours intramuscularly.
   6. Encourage cough, with breathing exercises.
   7. Daily or necessary thoracenteses without air replacement.
   8. Evacuate when transportable.

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EARLY PULMONARY DECORTICATION IN THE TREATMENT OF POSTTRAUMATIC EMPYEMA

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OF ALL THE TRAGIC SEQUELAE OF WAR, few are more distressing than the problems of those whose injuries result in chronic intrapleural sepsis. These unfortunates are inevitably found in large numbers through the post-bellum years either doggedly submitting to one major operative procedure after another, or resignedly suppurating through a shortened life-span of chronic invalidism.

Surgeons interested in thoracic disease have long been mindful of the magnitude of the problem of chronic empyema and have been prodigious in their efforts toward its solution. Despite many notable contributions elucidating many of the factors concerned in the inception of chronic empyema, it still occurs all too frequently following injury to the thorax. An examination of the voluminous literature on the subject brings out the striking fact that empyema is thought of and written about, for the most part, as though all empyemas were of the same generic type. Posttraumatic empyema certainly gives rise to a far higher incidence of chronicity than does meta- or post-pneumonic empyema. Yet little effort is made in the literature to establish vital, fundamental differences. The inference is too often left that empyema thoracis is empyema thoracis, regardless of its mode of origin. Yet significant differences do exist and must be recognized if we are to approach properly the posttraumatic empyema problem. That this difference was not more clearly recognized and emphasized by those who studied the large number of empyemas during World War I is remarkable. Perhaps the correct explanation is Churchill's[1] suggestion that the influenzal empyemas so overshadowed the posttraumatic cases in numbers and in interest that the individuality of the latter group was not recognized.

Despite modern surgical therapy and various chemotherapeutic adjuncts it now seems clear that no less than 15-20 per cent of those receiving penetrating and perforating thoracic wounds in this war will develop posttraumatic empyema[2]. When one contemplates the global scale of this conflict, and the probable total number of casualties, one becomes aware of the unprecedented chronic empyema potential that exists. The value of any method of treatment that will minimize the occurrence of chronic, crippling intrapleural infection following wounds of the chest is readily apparent.

This paper is a presentation of what we believe to be the most rational

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and effective method of treating posttraumatic empyema and represents the most productive approach, we believe, toward the prevention of the chronic phase of that disease. We are aware that much of this represents a radical departure from time-honored concepts.

In any study of posttraumatic empyema one is struck by the importance of the presence of blood in the pleural cavity as the antecedent factor. Much of the knowledge that we have gained pertaining to posttraumatic empyema has stemmed directly from a study of the problem of hemothorax. The vast majority of posttraumatic empyemas are infected hemothoraces, and the present rationale of treatment represents the direct application of those principles learned in dealing with hemothoraces both clotted and unclotted. Since

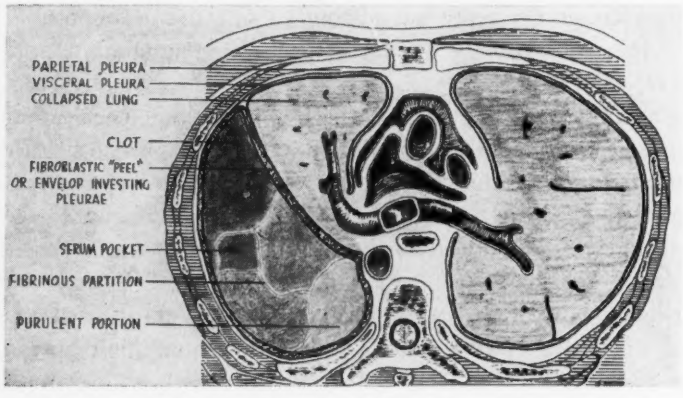

FIG. 1.—Photograph of diagram of cross-section of chest, with an infected organizing hemothorax of the left side.

the vast majority of posttraumatic empyemas develop on a basis of an hemothorax, the nature of the empyema can best be understood by an inquiry into the pathology of that entity. Studies of the intrapleural changes in hemothorax have been remarkable chiefly by their absence. The thoracoscopic examination of the pleura in cases of hemothorax by Edwards and Davies[3], in 1940, represents one of the first serious efforts to ascertain the nature of the pleural response to blood in the pleural cavity.

The opportunity to operate upon and decorticate the lung of a case of clotted, organizing hemothorax, by one of us (T. H. B.) during the African Campaign, led to a recognition of the essential pathology of hemothorax and to a study of a large group of similar cases. This experience has yielded much valuable and applicable information.

As has been pointed out in a recent paper by Samson, Burford, Brewer and Burbank[4] all hemothoraces are associated with very typical and definite changes within the pleural cavity of greater or lesser degree. The essential feature is the formation of a fibroblastic membrane or "peel" over the visceral and parietal pleurae. This membrane forms a sac or envelope within which the hemothorax is contained. The relationship of this hemothoracic envelope to the visceral and parietal pleurae can be readily visualized by consulting

Figure 1. This membrane forms from the deposition of fibrin on the pleural surfaces. Red blood cells are caught in the fibrin meshes and a definite "peel" very early takes form. This layer begins to undergo rapid active fibroplasia and angioplasia, and one can soon recognize an "older" side toward the pleura and a "younger" side toward the hemothorax. From the pleural side of the fibroblastic membrane, fibroblasts wander out toward the younger portion and nests of angioblasts rapidly give rise to capillaries. The transition to adult fibrous tissue is prompt, and by three weeks the cellular pattern of the "peel" is well defined. Figure 2 shows the cellular pattern of a three

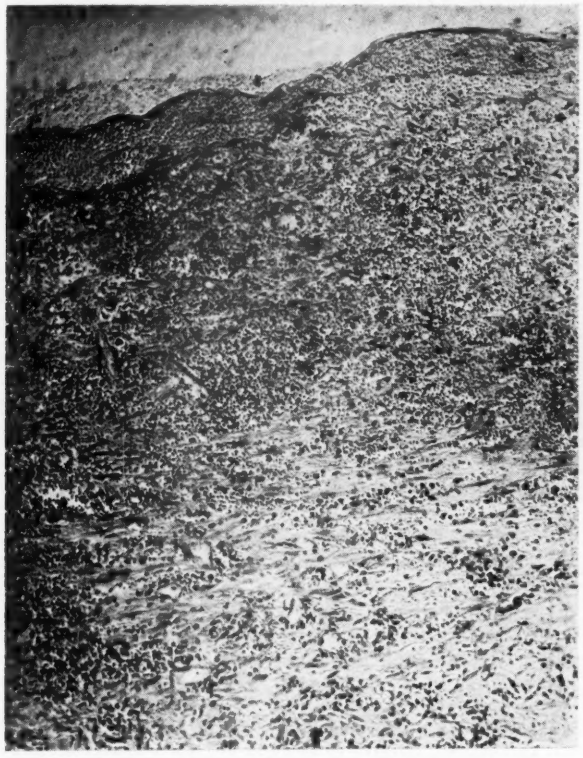

FIG. 2.—Photomicrograph of fibroblastic membrane of three weeks duration. Note active fibroplasia proceeding from pleural surface of "peel," which is at bottom of figure toward the "younger" portion at top of figure. (× 100)

week "peel," and shows the fibroblastic nature of the membrane. As the process grows older the cellular intimacy between membrane and pleura becomes greater until by eight or nine weeks the majority of cases will present definite symphysis. Until this time the pleura remains remarkably normal, and a cleavage plane between "peel" and pleura is easily established. Forming in the presence of a collapsed or partially collapsed lung, this membrane maintains the collapse of the lung and forms the chief deterrent to its reëxpansion. Figure 3 is a photograph of portion of a typical fibroblastic membrane removed from a case of posttraumatic empyema.

Hemothorax represents the largest hematoma with which the body has to deal. It is for the most part far too large to vascularize rapidly, and this fact may very well play a rôle in its tendency toward infection. This is particularly true of a clotted one which cannot be aspirated. In a liquid hemothorax prompt aspiration with pulmonary reëxpansion results in a cessation and a resolution of the changes described. If infection supervenes in either the liquid or the clotted hemothorax the resulting empyema will be unique in two respects. First, the lung is collapsed and compressed to a greater or lesser

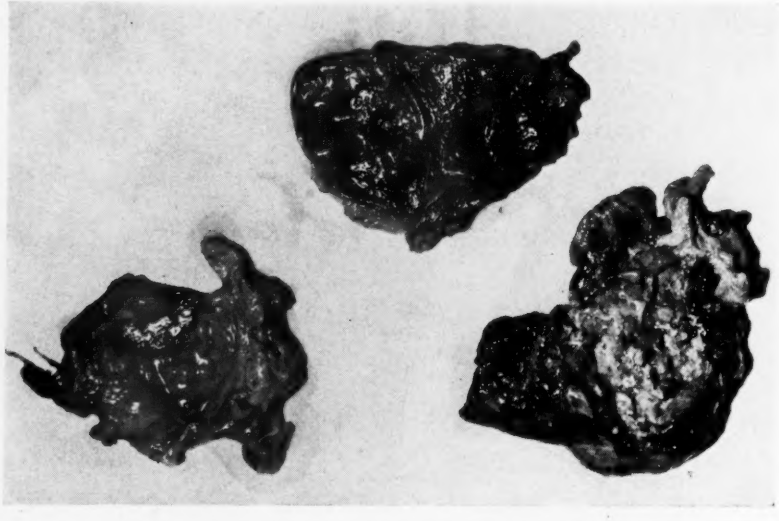

FIG. 3.—Photograph showing gross appearance of three-week-old fibroblastic membrane, or "peel," removed from visceral pleura of a case of empyema supervening upon an organizing hemothorax.

degree by the intrapleural blood even before infection occurs. Second, there rapidly develops, as described above, a constrictive pulmonary investment which restrains pulmonary reëxpansion. The recognition of these two factors is of prime importance in formulating a rational effective therapeutic approach to the problem of the empyema of trauma. Such a concept embodies not only evacuation of the pleural contents but also pleural divestment which will result in immediate and total primary pulmonary reëxpansion and complete obliteration of all pleural "dead space."

Fowler,[5] in 1893, and Delorme,[6] independently, in 1894, were the first seriously to attempt pulmonary mobilization in chronic empyema, by decortication. That the operation was so rarely successful was due to the fact that it was not attempted until late in the disease when, as we have seen from the discussion above, a complete fusion existed between pleura and the fibroblastic membrane. That the original attempts to perform decortication were made only upon patients who had been infected for many weeks, and often months, is understandable. To operate in the presence of pus without the advantages of bacteriostasis would have been inadvisable. These workers

were forced to adequately drain the empyema and await partial cavity sterilization. The idea of operating upon the acute phase of empyema was not to be considered in that time. Hedblom's[7] series of 30 cases, with 20 cures and one operative death, was the best reported but was not equaled by others. Decortication, except for the isolated instance, fell into disrepute and procedures designed to collapse the chest wall in upon the lung and thus obliterate the empyema cavity were devised. These procedures were admittedly serious compromises with the ideal and in no sense were restorative of function.

The operations of Schede,[8] Estlander,[9] the discussion procedure of Ransohoff,[10] and various combinations of these with partial decortications, such as the operation of Keller,[11] became the accepted operative approach to chronic empyema. The operative mortality remained high and the road to a cure for the patient remained a long hard one.

Samson and Burford (*loc. cit.*) in the course of a rather extensive experience in the decortication of large organizing hemothoraces were able to study a group of 15 early posttraumatic empyemas treated by decortication. Part of these were found to be infected at operation, when locules of pus were found within the hemothoracic cavity (see Figure 1) and when the case had been submitted to operation in the belief that a noninfected organizing hemothorax was being dealt with. A few were purposely operated upon immediately after obtaining positive smears or positive cultures upon examination of the fluid obtained by thoracentesis. The remainder were known massive empyemas submitted to decortication after preliminary rib resection drainage with attempts to sterilize the cavity. No case of known, frankly purulent, posttraumatic empyema was submitted to primary thoracotomy with decortication. The results in this group were encouraging. Of the 15, five obtained a prompt cure, five were markedly improved but retained rather small basal empyemas, and in the remaining five, large empyemas persisted but the cavity was smaller than the original process.

The advent of penicillin into this Theater and the contributions of Lyons,[12] which have so significantly influenced the handling of wound sepsis in general, stimulated the present study.

Penicillin, with its known property of bacteriostasis and, thus, of diminishing the danger of invasive infection when operating in the presence of acute infection, seemed to us to be the awaited, necessary adjunct to render safe the early application of more effective methods to the therapy of pleural sepsis. The pursuance of a rational plan of treatment in posttraumatic empyema directed toward achieving early pleuropulmonary lysis, total ablation of infected tissue, complete primary pulmonary reëxpansion, with absolute obliteration of residual pleural "dead-space" relatively free from the danger of septic dissemination, at last seemed safe and applicable.

DESCRIPTION OF CLINICAL MATERIAL

In March, 1944, we began subjecting all suitable cases of posttraumatic

empyema to thoracotomy, with decortication after an initial period of penicillin preparation. Twenty-five such cases have been treated by this method since then. The majority of the cases in this series have been treated at a Thoracic Center in the North African Theater of Operations. This paper represents an account of that experience. All cases in this group were definite empyemas. In two, however, there was failure to obtain positive cultures from the pleural pus. This is not a higher incidence of failure to obtain growth from pus than one would encounter in any series of 25 cases of empyema, particularly since the use of the sulfonamides and penicillin is so widespread. All had clinical evidence of infection: *viz.*, fever, anorexia, malaise and the clinical appearance of toxicity. The majority were acutely and severely ill. At operation, all had definite pus in the pleural cavity.

The selection of cases was in the direction of leaving the less severe ones to be handled by simple rib resection drainage, and choosing those with the more complete types of empyema; *i.e.*, those most likely to develop chronic empyema. If the empyema was total, or if the apex was involved in the collapse, the case was selected for thoracotomy with decortication. Sizeable (above 1.5 cm. in greatest diameter) intrapulmonary foreign bodies were considered important reasons to perform thoracotomy-decortication procedures when the empyema was less than total. Intrapleural foreign bodies were considered an even more important indication for operation. Failure of the lung to reexpand progressively and quickly (within four weeks) after adequate rib resection drainage was also considered a most important indication to open the chest and decorticate the lung.

Though we feel that thoracotomy with decortication without preliminary drainage to be the method of choice a few cases in any series will be too sick initially to withstand major surgery. In these, of course, a preliminary rib resection drainage will be necessary. Eight of the 25 cases in this series presented this type of problem.

For the small basal empyemas as well as for the smaller encapsulated empyemas in other regions of the thorax we believe properly placed drainage to be the method of choice. These seldom give rise to persistent cavities if properly drained.

All the cases were males between the ages of 18 and 30.

The essential data on these 25 cases is summarized in Table I. Complete case records of Cases 6 and 22 are included, and are representative of the series.

REPRESENTATIVE CASE RECORDS

Case 6.—This patient sustained severe multiple penetrating shell fragment wounds of the right thorax, right arm, and right thigh on March 26, 1944. First aid was administered immediately by the Company Aid Man, and the patient promptly moved to the Aid Station, where plasma and morphia were given. Within 12 hours of wounding he was admitted to a Forward Evacuation Hospital, where all wounds were débrided and the patient transfused. The patient was again wounded when the hospital

was bombed, suffering a severe laceration of the scalp from a bomb fragment. This wound was débrided and sutured. Signs of right hemothorax were increasingly apparent and a thoracentesis was performed March 31, 1944, which yielded 1,100 cc. of serosanguineous fluid. Thoracenteses were repeated on April 1 and April 5, yielding 1,050 cc. and 1,000 cc. of similar fluid, respectively. On April 9, patient was admitted to the Chest Center in the Base. Examination revealed a moderately febrile patient, who appeared acutely ill but in no significant respiratory distress. Signs of intrapleural fluid were present on the right. A healing scalp wound and an infected wound of the right thigh were present. The débrided wounds of the right arm and right thorax were clean and granulating. A thoracentesis, performed April 9, returned 750 cc. of dark bloody fluid with an offensive odor. Cultures were made of this fluid. Six hundred fifty cubic centimeters of similar fluid were removed on the following day and on April 11, 625 cc. were obtained. On April 12, cultures of the previously obtained pleural fluid were reported positive for *Clostridia*. Systemic penicillin was begun on this date, the patient being given 25,000 units intramuscularly every three hours. The temperature at this time was running from 101° to 102° F., rectally. The patient appeared mildly toxic and was anorexic. On April 13, the fluid from the right chest was even more offensive and showed early purulent transition. A roentgenogram of the chest showed a marked intrapleural process on the right side, with considerable pulmonary collapse, the apex being compressed to the level of the fifth rib posteriorly. An intrapleural metallic foreign body could be visualized (see Figure 7A). The institution of penicillin therapy did not result in any reduction in fever but did seem to bear favorably upon the patient's appetite and general condition. Specimens of pleural fluid continued to become progressively more purulent, and the organism was identified as a definite pathogenic proteolytic *Clostridium*. Repeated blood transfusions were given, bringing the patient's red blood cell volume up to 40. On April 18, 1944, 23 days after injury, and after six days on penicillin protection, immediately following excision and suture of the wound of entry, a thoracotomy with evacuation of the pus, and decortication of the lung was performed. A right posterolateral incision was made and the pleural cavity entered through the sixth interspace. A large hemothoracic envelop was found containing a large amount of very foul-smelling pus, liquefying blood clot and fibrin. A metallic foreign body was found lying free in the empyema cavity. No fistulae were present. The lung was collapsed an estimated 75 per cent. The pus was aspirated, the cavity cleansed with saline irrigations, and a line of cleavage readily found between the visceral pleura and the fibroblastic membrane. The latter measured 4 mm. in thickness (see Fig. 3). This membrane stripped readily from the pleura and the entire lung decorticated easily. After decortication, the visceral pleura presented an entirely normal appearance. Once this has been accomplished the lung reëxpanded completely, filling the chest and obliterating all pleural "dead space." No leaks were present in the lung. The pleural cavity was again irrigated with physiologic saline solution and an internal intercostal nerve block with 1 per cent novocaine done. Anterior and posterior intercostal "water-seal" catheters were placed, the former in the second interspace in the midclavicular line, and the latter in the eighth interspace in the posterior axillary line. The chest was then closed, using silk throughout. Twenty-five thousand units of penicillin in 100 cc. of sterile water were injected into the chest through the catheters and the catheters clamped. Fifteen hundred cubic centimeters of whole blood were given during the operation. The patient was bronchoscoped at the completion of the operation. Following operation the posterior catheter was left clamped for six hours to allow the penicillin to remain within the pleural space. The anterior catheter was connected to a "water-seal" bottle to permit the egress of trapped air.

The postoperative course was entirely uneventful. Defervescence was prompt, the temperature reaching normal on the fifth day, and remaining so. The anterior catheter was removed on the second postoperative day. The wound healed without infection, and

the sutures were removed on the seventh postoperative day, at which time the patient was allowed up. Penicillin was discontinued on the eighth postoperative day. The patient's convalescence was rapid and a roentgenogram made three weeks after operation showed a clear chest, with a completely expanded lung (see Fig. 7B). He was discharged to full duty, completely cured on June 5, 1944, only 70 days after injury. We have since learned that he accompanied his combat outfit in a recent amphibious operation.

Case 22.—This patient suffered a penetrating shell fragment wound of the left thorax on June 1, 1944. He was given almost immediate resuscitation and within 20 hours of injury had been admitted to a Forward Evacuation Hospital, where intercostal nerve block, thoracentesis and débridement of the wound of entry was done. Thoracentesis on June 2 was productive of 300 cc. of bloody fluid and, on June 3, 750 cc. of similar fluid were removed. On June 5, 500 cc. were obtained, and at this time it was noted that the fluid had an early purulent character. The patient was admitted to the Chest Center June 6, 1944. Upon admission, he was acutely ill, slightly cyanotic and moderately dyspneic. The temperature was elevated, and patient appeared toxic. Roentgenograms of the chest showed an extensive left-sided intrapleural process, with apical collapse, and an intrapulmonary metallic foreign body (see Fig. 12A). Thoracentesis yielded 250 cc. of thin sanguino-purulent material, with a foul odor. A culture of this was positive for proteolytic *Clostridia*. On June 7, 1944, systemic penicillin was started, 25,000 units being given every three hours. Daily blood transfusions of 500 to 1,000 cc. were given. Thoracotomy with decortication was performed June 8, 1944, one week after injury. A left posterolateral incision was made and the pleural cavity entered through the sixth interspace. A large empyema cavity was found. This was filled with thin, malodorous pus. A fistula, two centimeters in diameter, was present in the base of the upper lobe at the fissural margin. Investigation of the fistulous opening showed it to communicate directly with a pulmonary abscess, four centimeters in diameter, with the lung in the collapsed state. The metallic foreign body, an irregular piece of shell casing, 1.9 cm. in its largest diameter, was found lying free in the abscess cavity. The empyema cavity was aspirated free of pus and fibrin masses, and the "peel" decorticated from the visceral pleura. The "peel" stripped off readily, leaving a normal glistening visceral pleura. The lung reëxpanded to fill the hemothorax. A laceration extending into the abscess, presenting a fistula in the upper lobe, was enlarged and the foreign body removed. The lining of the cavity was débrided by sharp dissection down to normal lung tissue. The defect in the lung was then closed in layers. Intercostal nerve block with novocaine of nerves three to nine, inclusive, was done, and the anterior and posterior intercostal catheters placed, as described in Case 6. The chest was closed in layers, using fine, interrupted silk sutures. Twenty-five thousand units of penicillin in 100 cc. of sterile water were introduced into the pleural cavity through the catheters and the catheters clamped. The patient was bronchoscoped at the conclusion of the operation. Fifteen hundred cubic centimeters of blood were given during the procedure. On being returned to bed the anterior tube was connected to a "water-seal" bottle, the posterior tube being left clamped for six hours. Despite the extensive operative procedure the patient manifested very little postoperative reaction and the temperature and pulse returned to normal on the seventh postoperative day (see Fig. 6). The temperature rise on the ninth postoperative day corresponded to the removal of the posterior intercostal tube. The anterior catheter had been removed on the third postoperative day. The temperature and pulse remained normal, and the wound healed without infection. The patient's return to normal activity was rapid. A roentgenogram three weeks after operation showed a completely expanded lung and no intrapleural abnormalities (see Fig. 12B). The patient was discharged to full duty on July 28, 1944, eight weeks after injury, and participated in an amphibious operation within a short time after discharge.

PREOPERATIVE MANAGEMENT

Once the diagnosis of infection within the pleural space was established it was necessary to decide whether or not the case was one that could be handled best by thoracotomy with decortication, or whether simple rib resection drainage was the procedure of choice. It is our definite feeling that it is wisest to perform simple rib resection drainage in those cases where the pleural infection is limited to the basal portion of the thorax or where it is small and encapsulated. A few cases will be too sick initially to subject to the more radical procedure of open thoracotomy, with decortication, and in these a preliminary rib resection and drainage will be necessary. If cavity obliteration and pulmonary reëxpansion was not prompt and satisfactory the case then became a candidate for decortication. Needless to say, proper roentgenologic and laboratory studies had been made to establish an accurate picture of the patient's general condition with reference to pulmonary status, total protein and hematocrit. Studies of the pleural fluid identified the offending micro-organism, and the roentgenograms disclosed the presence of intrathoracic foreign bodies, gave evidence of bronchopleural fistulae, or other lesions.

As soon as evidence of intrapleural infection had been obtained, penicillin therapy was instituted, giving 25,000 units of penicillin every three hours—a total of 200,000 units every 24 hours. Daily transfusions of 500 to 1,000 cc. of whole citrated blood were given until the plasma proteins were restored to a normal level, and until the hematocrit values reached a normal of 40 per cent, or slightly above. The length of time that penicillin was given prior to operation was not uniform. Some patients did well when only 24 hours elapsed from the institution of penicillin therapy until operation. Others, for one reason or another, have been kept on the drug for as long as 17 days before being submitted to operation. It would seem that 48 to 72 hours would be a reasonable and rational period of preoperative penicillin therapy, before undertaking thoracotomy with decortication. Experience has taught that it is useless to await a defervescent phenomenon. That comes only with extirpation of the infected focus.

OPERATIVE TREATMENT

Endotracheal gas-oxygen-ether anesthesia was used in all cases. Unhealed wounds of the affected side were either closed secondarily, or excised and closed, using a separate set of instruments. If the wounds were too badly infected to be closed, or to be excised and closed, they were carefully isolated from the field of incision. In cases where a previous operation for drainage had been performed the drainage site was isolated and dealt with at the time of closure.

A posterolateral incision was made on the affected side just below the angle of the scapula, and the pleural cavity was entered through the sixth interspace in the majority of cases. Occasionally the fifth or the seventh

interspace was chosen, depending somewhat upon the thoracic habitus of the patient but more particularly upon whether one anticipated having to do more at the upper pole or more at the lower pole of the involved hemithorax. The great majority of the operations were performed without costal section or resection, although, in a few, previous operations or comminuted fractures of the ribs made it wise to resect all or part of a rib. Exposure is entirely adequate without costal mutilation and intercostal incision is the method of choice. Upon entering the pleural cavity it is extremely important to free the lung rather widely in all directions from the parietal pleural "peel" if it is adherent in the vicinity of the opening into the chest. If this precaution is not taken extensive damage will befall the lung when the ribs are spread. Since the "peel" on the parietal pleura is to be left it is important to bear in mind during this process of para-incisional mobilization that the proper line of cleavage lies between the two layers of fibroblastic membrane, *i.e.*, the one on the visceral pleura, and the other on the parietal, and not between either pleura and its respective "peel." This relationship can be readily visualized by an examination of Figure 1, where these layers are diagrammatically illustrated. Once this maneuver has been accomplished, rib spreading retractors are introduced and the ribs gradually spread to give exposure. It is important to widen the blades of the retractors gradually to allow for muscular relaxation so that fractures of the ribs, muscular avulsion or pleural tears will not occur.

Pus, clot fragments, and fibrin masses are cleansed out of the empyema cavity and the cavity examined for bronchopleural fistulae. If none exist it is well at this point to thoroughly wash out the empyema cavity with sterile physiologic saline. Decortication of the fibrino-fibrous investment on the visceral pleura is then begun by carefully incising it down to the visceral pleura. This maneuver is materially aided by having the lung "braced," as it were, against the incision by moderate positive pressure inflation. The moment the "peel" is completely disrupted in any segment the underlying lung will, if under positive pressure, immediately herniate through the incision, thus, accurately delineating the desired cleavage plane. The edge of the membrane is then grasped with forceps and the actual decortication begun. This is best done by gently and carefully dissecting with a small, firmly packed gauze "pusher." Particular care not to tear the lung must be exercised at the fissural margins and around fistulae, or healed areas that represent points of entrance or exit of a missile. When the visceral pleura has been completely decorticated the lung should be brought to full re-expansion and the surface carefully examined for tears in the visceral pleura and/or parenchyma. These, if present, are repaired by interrupted sutures of fine silk. Fistulae, if present, are best treated by freshening their margins and closing them with sutures of fine silk.

On three occasions in this series it has been necessary to deal with pulmonary abscesses at the time of operation (Cases 7, 21 and 22). We have elected to open the abscess completely, débride the necrotic lining membrane

by sharp dissection and close the defect in the lung with two or more layers of fine silk in such a fashion that all dead space was obliterated.

It will occasionally be found that after what may be termed the "primary" peel has been removed there will remain small scattered islands of a very thin, but surprisingly tough secondary membrane still adherent to the visceral pleura. These will be observed to be causing creasings and infoldings of the lung that materially hampers 100 per cent reëxpansion, and must be meticulously removed.

If an intrapulmonary foreign body is present of such a size as to warrant removal, there should be no hesitancy in opening the lung, extracting the missile and repairing the lung with fine silk.

No attempt is made to remove the membrane from the parietal pleura. It is mandatory, however, to see that a completely smooth margin is left around the entire circumference of the reflection of the peel from the parietal to the visceral pleura. Cuffs and strands of the unattached membrane can only serve as potential sites for the pocketing of secretions.

The diaphragm will be found almost universally elevated and fixed. While there is no need to decorticate it, yet it is felt wise to free this structure circumferentially and thus mobilize it. Note is made of the costophrenic and cardiophrenic sinuses to see that they are free of clot and membrane.

The entire internal chest wall is inspected and palpated for the presence of rib splinters, or metallic foreign bodies which partially protrude into the chest. If present, these are removed and a smooth surface achieved.

Every effort is directed during the entire procedure to achieve complete pulmonary reëxpansion and complete obliteration of intrapleural "dead-space." Any compromise with this ideal is a certain step toward failure. The operation that ends failing to bring the lung into absolute contiguity with the chest wall, and/or that fails to make certain that it remains there has failed to achieve its purpose, and has little likelihood of succeeding. It is a fallacy to assume that half measures in this direction will, by the generous intervention of providence, or something equally unlikely, result in a cure.

Provision for the maintenance of pulmonary expansion is made by the insertion of two, and frequently three intercostal tubes which are subsequently connected to "water-seal" bottles. One of these is placed in the eighth interspace in the posterior axillary line. This tube is a fenestrated piece of soft rubber clysis tubing of 10-mm. internal diameter. It is carried into the pleural space for a distance of two and one-half to three inches, and its proximal end tacked to the parietal pleural "peel" with one silk suture. For the second tube a small Pezzar catheter (No. 12 or 14) is used, the tip being cut off so that just a flange remains. This is brought out through the second interspace anteriorly in the midclavicular line. If the third tube is employed, it is of the same type as the second, and is brought out through either the fifth or sixth interspace in the midclavicular line. The tubes are clamped temporarily. It may be pointed out here that tubes within the pleural cavity are not, and do not, act as foreign bodies *per se*. They only become that

when they cease functioning in their all-important capacity of maintaining pulmonary expansion, and obliteration of pleural "dead-space." When they cease to facilitate progress in that direction, then, and only then, do they become foreign bodies, and should be removed promptly. If the case has been one in which a previous rib resection for pleural drainage has been performed it is altogether likely that the drainage site will correspond with the site of election for the posterior tube. In these cases we have excised the margins of the wound for preliminary drainage, placed the tube through it, and sutured about the tube so that it was air-tight. Any coincident chest wall defect must be freshened and closed solidly.

Intercostal nerve block with 1 or 2 per cent novocaine is done at this time, blocking two or three nerves above the interspace of entrance and two or three below.

A final copious lavaging of the pleural cavity with sterile physiologic saline is carried out, and the cavity aspirated completely dry. A final inspection is made to assure that a completely clean field is being left. The lung is then brought gradually to full expansion. When the lung is completely out to the chest wall the closure is proceeded with. We have used interrupted silk sutures exclusively. Pericostal sutures are not used.

When the closure has been completed 25,000 units (occasionally 50,000) of penicillin in 100 cc. of physiologic saline are injected into the cavity through the tubes, an equal amount being allocated to each tube. The one or two anterior tubes are immediately allowed to drain under water, but the posterior tube is left occluded for six hours following operation to prevent the escape of the penicillin solution. The patient is bronchoscoped before leaving the table.

Blood is given during the operation, a total of 1,000 to 2,000 cc. being administered during the procedure. Having brought the patient to the operating room with a normal hematocrit it is just as important to have him enter the reparative phase of his course with the advantage of a full compliment of red blood cells, hemoglobin, and plasma protein. This aspect is no less important than the shock-prevention function of blood replacement in the end-result.

POSTOPERATIVE MANAGEMENT

Following the return of the patient to the ward he is placed on his back with the bed level. As soon as the patient has fully reacted, the back rest is elevated and the patient is instructed to breathe deeply and cough at intervals. Morphine is administered as required. Food is permitted, as tolerated, and intravenous fluids are given as indicated. One or two transfusions during the first two or three postoperative days are usually given, depending upon the hematocrit. Systemic penicillin is continued until the tubes are out and the patient has been afebrile for two or three days. The tubes are checked frequently to assure their proper functioning. The anterior catheter is removed as soon as apical expansion has been obtained, and there is no

further oscillation of the water column on cough. This is usually a matter of 48 to 72 hours. The posterior tube is left as long as there is any serous drainage through it. This will vary rather widely. The majority may be safely removed between the seventh and tenth postoperative days.

In the event that a complete cure does not occur, and the patient develops a basal empyema, a two-inch section of rib is removed (usually the eighth) in the posterior axillary line and a large rubber tube introduced and made air-tight. This is connected to a "water-seal" bottle. Procrastination in providing adequate drainage, once a recurrence of the empyema has occurred will inevitably result in a more extensive cavity forming than if proper drainage is promptly instituted.

Sutures are removed on the sixth or seventh day and the patient allowed up. A return to full activity is encouraged as rapidly as is consistent with the patient's strength. Deep breathing and postural consciousness are stressed. Shoulder and arm motion of the operated side are encouraged very early in the postoperative course.

RESULTS

Nineteen of 25 cases obtained primary cure of the empyema. Six of the 25 developed a small recurrent basal empyema. The only deviation from 100 per cent *per primam* wound healing in the entire series was a trivial superficial wound infection which occurred in Case 1. The fever in all instances subsided promptly. Postoperative reactions were remarkably mild. No case developed any evidence of embolic infection during the period of time they were under our observation. To date we have no knowledge of the occurrence of any such complication in any case. The one death in the series occurred eight weeks postoperatively, and cannot be considered an operative death (Case 25).

COMPLICATIONS

Complications have been gratifyingly few. In the 25 cases all wounds healed solidly and *per primam*. In only one case (Case 1) was there any wound sepsis. This was a simple superficial wound infection which cleared promptly. In six instances there was a basal recurrence of the empyema requiring secondary thoracotomy with rib resection. In all, the empyema was much less than the original process and in only one patient is there any likelihood that a further obliterative procedure will need to be undertaken to effect a cure. The one death in the series presented a recurrent empyema, recurrence of multiple bronchopleural fistulae, and bronchopneumonia of the contralateral lung. In Case 21, there was reopening of the abscess and a recurrence of the empyema. This remained localized to the apex and was very much less in extent than the involvement at thoracotomy.

DISCUSSION

We feel that the valid applicability of early reparative surgical measures to the problem of traumatic pleural infection has been demonstrated. In addition to the importance of the early cure of the infection, the complete restoration of pulmonary function as rapidly as possible is clearly of vital importance. Before the present concept could be evolved and applied it was first necessary to demonstrate certain fundamentals. The first of these, the essential nature of the pathology of posttraumatic empyema, grew out of a study of the pathology of hemothorax. The demonstration of the fibroblastic membrane investing the pleurae was tantamount to an understanding of most of the problems involved in the treatment of posttraumatic empyema. This discovery led to the realization of what must be accomplished if a prompt cure was to be obtained. It, likewise, explained many of the cases of chronicity and clearly pointed the course to their prophylaxis. The demonstration of the efficacy of penicillin to control bacterial invasiveness and to thus render surgery safely applicable at the optimum time to obtain the maximal functional result (Lyons, *loc. cit.*), was the second of these fundamentals.

In this series of 25 cases of posttraumatic empyema treated by thoracotomy with decortication, under penicillin protection, there has been no favorable selection of cases. The cases have been representative (Table I). All were clinically ill. All had definite empyemas. In the main, they represent the more severe and complicated types. This was inevitable, since as pointed out in the discussion of the selection of cases, those with only basal or small encapsulated empyemas were treated by rib resection drainage alone. The fact that three cases of associated pulmonary abscess were encountered in the series will demonstrate the fact that favorable cases were not selected. The term pulmonary abscess as used here perhaps requires further description. The abscesses represented, in fact, seriously infected lacerations with cavity formation and suppuration.

An examination of Table I will reveal that the series may be divided readily into two groups. The first group of 18 cases presented only pleural involvement. The second group (seven cases) had in addition to pleural infection, significant pulmonic pathology including three cases of pulmonary abscess associated with metallic foreign bodies, bone fragments, and clothing. This distinction is important in an analysis of the results. Of the 18 cases having no significant pulmonic pathology there were 16 cases of primary cure. The use of the word "cure" indicates that the lung fully expanded and completely obliterated the pleural cavity, and that there was no clinical or roentgenographic evidence of a persistence or recurrence of the empyema during the period of postoperative observation varying from six to ten weeks. In all of the 19 cases classed as cured, the complete obliteration of the pleural space and the disappearance of evidence of infection had taken place within two weeks after operation. Of the two in this group that developed a re-

TABLE I

SUMMARIZED DATA OF CASES

Case No.	Type of Injury	Time from Admission to Base Hospital to Operation	Time from Injury to Operation in Days	Type of Hemothorax	Etiologic Organism of Empyema	Previous Surgical Therapy	Associated Thoracic Pathology at Time of Definitive Therapy	Definitive Therapy	Result
1.	SFW pen. left	26 days	28 days	Liquid	*Hemolytic Staphylococcus*	Débridement. Thoracentesis. Secondary closure	Metallic foreign body, left lower lobe.	Penicillin protection 24 hours. Thoracotomy with decortication. Local penicillin. Penicillin 11 days postoperative	Superficial localized wound infection cleared promptly. Complete primary cure.
2.	SFW pen. right	10 days	35 days	Clotted	*Hemolytic Staphylococcus aureus*	Débridement. Thoracentesis. Celiotomy. Seondary closure	Metallic foreign body, right lower lobe	Penicillin protection 24 hours. Thoracotomy with decortication, removal metallic foreign body from lung. Local penicillin. Penicillin 10 days postoperative	Complete primary cure. No complications.
3.	SFW pen. left	30 days	28 days	Clotted	*Hemolytic Staphylococcus albus*	Débridement. Thoracentesis. Secondary closure	Metallic foreign body, left lower lobe. Comminuted fracture 7th rib left	Penicillin protection 24 hours. Thoracotomy with decortication, removal metallic foreign body from lung. Local penicillin. Penicillin 10 days postoperative	Complete primary cure. No complications.
4.	SFW perf. right	32 days	42 days	Liquid	*Hemolytic Streptococcus*	Débridement. Thoracentesis. Rib resection drainage. Secondary closure	None	Penicillin protection 48 hours. Thoracotomy with decortication. Local penicillin. Penicillin 12 days postoperative	Complete primary cure. No complications.
5.	SFW pen. right	34 days	42 days	Liquid	*Micro-aerophilic Streptococcus*	Débridement. Thoracentesis. Rib resection drainage. Secondary closure	Metallic foreign body, right lower lobe	Penicillin protection 48 hours. Thoracotomy with decortication, removal metallic foreign body from lung. Local penicillin. Penicillin 10 days postoperative	Complete primary cure. No complications.

TABLE I—(*Cont.*)

SUMMARIZED DATA OF CASES

Case No.	Type of Injury	Time from Admission to Base Hospital to Operation	Time from Injury to Operation in Days	Type of Hemothorax	Etiologic Organism of Empyema	Previous Surgical Therapy	Associated Thoracic Pathology at Time of Definitive Therapy	Definitive Therapy	Result
6.	SFW pen. right	9 days	21 days	Clotted	*Proteolytic Clostridia*	Débridement. Thoracentesis. Secondary closure	Intrapleural metallic foreign body	Penicillin protection 6 days. Thoracotomy with decortication, removal intrapleural metallic foreign body. Local penicillin. Penicillin 8 days postoperative	Complete primary cure No complications.
7.	SFW pen. left	16 days	16 days	Liquid	*Nonhemolytic Streptococcus*	Débridement. Thoracentesis. Secondary closure	Metallic foreign body, left lower lobe. Bronchopleural fistulae	Penicillin protection 24 hours. Thoracotomy with decortication, removal metallic foreign body from lung. Closure bronchopleural fistulae. Local penicillin. Penicillin 13 days postoperative	Complete primary cure. No complications.
8.	SFW pen. right	6 days	10 days	Clotted	Definite pus. No organism identified	Débridement. Thoracentesis. Secondary closure	Metallic foreign body, right lower lobe	Penicillin protection 6 days. Thoracotomy with decortication, removal metallic foreign body from lung. Local penicillin. Penicillin 6 days postoperative	Complete primary cure No complications.
9.	SFW pen. left	16 days	19 days	Clotted	*Proteolytic Clostridia*	Débridement. Thoracentesis. Secondary closure	Metallic foreign body, left upper lobe	Penicillin protection 24 hours. Thoracotomy with decortication, removal metallic foreign body from lung. Local penicillin. Penicillin 12 days postoperative	Complete primary cure. No complications.
10.	SFW pen. right	32 days	21 days	Clotted	Definite pus. No organism identified	Débridement. Thoracentesis. Early thoracotomy E. H. Secondary closure	Metallic foreign body, right lower lobe	Penicillin protection 24 hours. Thoracotomy with decortication, removal metallic foreign body from lung. Local penicillin. Penicillin 7 days postoperative	Complete primary cure. No complications.

TABLE I—(*Cont.*)

SUMMARIZED DATA OF CASES

Case No.	Type of Injury	Time from Admission to Base Hospital to Operation	Time from Injury to Operation in Days	Type of Hemothorax	Etiologic Organism of Empyema	Previous Surgical Therapy	Associated Thoracic Pathology at Time of Definitive Therapy	Definitive Therapy	Result
11.	GSW pen. left	3 days	11 days	Liquid	*Proteolytic Clostridia*	Early thoracotomy E. H. Thoracentesis. Secondary closure	None	Penicillin protection 48 hours. Thoracotomy with decortication. Local penicillin. Penicillin 14 days postoperative	Complete primary cure. No complications.
12.	Crush injury to chest	5 days	31 days	Clotted	*Proteolytic Clostridia. Hemolytic Staphylococcus aureus*	Thoracentesis	None	Penicillin protection 48 hours. Thoracotomy with decortication. Local penicillin. Penicillin 10 days postoperative	Complete primary cure No complications.
13.	GSW perf. left	4 days	50 days	Clotted	*Hemolytic Staphylococcus aureus*	Débridement. Thoracentesis. Secondary closure	None	Penicillin protection 24 hours. Thoracotomy with decortication. Local penicillin. Penicillin 13 days postoperative	Small basal empyema requiring rib resection drainage.
14.	SFW pen. left	27 days	36 days	Clotted	*Hemolytic Staphylococcus aureus*	Early thoracotomy E. H.	Metallic foreign body embedded in posterior parietal pleura	Penicillin protection 48 hours. Thoracotomy with decortication. Local penicillin. Penicillin 8 days postoperative	Complete primary cure. No complications.
15.	SFW perf. right	20 days	28 days	Clotted	*Proteolytic Clostridia*	Débridement. Closure sucking wound. Thoracentesis. Secondary closure	None	Penicillin protection 48 hours. Thoracotomy with decortication. Local penicillin. Penicillin 8 days postoperative	Developed intra-abdominal abscess, associated with intra - abdominal foreign body. Primary cure of empyema.
16.	GSW pen. right	30 days	33 days	Clotted	*Proteolytic Clostridia*	Early thoracotomy E. H.	Intrapulmonary foreign body	Penicillin protection 24 hours. Thoracotomy with decortication. Local penicillin. Penicillin 7 days postoperative	Complete primary cure No complications.

TABLE I—(*Cont.*)

SUMMARIZED DATA OF CASES

Case No.	Type of Injury	Time from Admission to Base Hospital to Operation	Time from Injury to Operation in Days	Type of Hemothorax	Etiologic Organism of Empyema	Previous Surgical Therapy	Associated Thoracic Pathology at Time of Definitive Therapy	Definitive Therapy	Result
17.	SFW perf. right	55 days	64 days	Liquid	*Anaerobic Streptococcus hemolytic. Staphylococcus aureus*	Early thoracotomy E. H. Rib resection drainage for empyema	None	Penicillin protection 48 hours Thoracotomy with decortication. Local penicillin. Penicillin 15 days postoperative	Complete primary cure. No complications.
18.	SFW pen. right	18 days	20 days	Clotted	*B. proteus*	Débridement. Thoracentesis. Secondary closure. Reduction compound fracture right femur. Application skeletal traction	None	Penicillin protection 5 days. Thoracotomy with decortication. Local penicillin. Penicillin 16 days postoperative	Basal empyema requiring rib resection drainage. Empyema cleared after secondary drainage. Skeletal traction of femur probably influenced development of recurrent empyema.
19.	SFW perf. right	69 days	78 days	Liquid	*Anaerobic Streptococcus. Sporogones*	Early thoracotomy E. H. Anterior and posterior rib resection drainage for empyema	Multiple bronchopleural fistulae	Penicillin protection 48 hours Thoracotomy with decortication. Local penicillin. Penicillin 8 days postoperative	Complete primary cure. No complications.
20.	SFW pen. left	17 days	42 days	Liquid	*Anaerobic Streptococcus*	Early thoracotomy E. H. Anterior and posterior rib resection drainage for empyema	Multiple bronchopleural fistulae	Penicillin protection 72 hours. Thoracotomy with decortication. Local penicillin. Penicillin 13 days postoperative	Complete primary cure. No complications.
21	SFW pen. right	10 days	14 days	Clotted	*Proteolytic Clostridia*	Débridement. Thoracentesis Secondary closure	Large intrapulmonary abscess right upper lobe, associated with large intrapulmonary foreign body. Bronchopleural fistulae	Penicillin protection 5 days. Thoracotomy with decortication. Débridement closure of abscess. Local penicillin. Penicillin 14 days postoperative	Partial reopening of abscess. Recurrence of apical empyema requiring rib resection drainage. Recurrent empyema less than original process. Progress satisfactory.

TABLE I—(*Cont.*)

SUMMARIZED DATA OF CASES

Case No.	Type of Injury	Time from Admission to Base Hospital to Operation	Time from Injury to Operation in Days	Type of Hemothorax	Etiologic Organism of Empyema	Previous Surgical Therapy	Associated Thoracic Pathology at Time of Definitive Therapy	Definitive Therapy	Result
22.	SFW pen. left	2 days	8 days	Clotted	*Proteolytic Clostridia*	Débridement. Thoracentesis. Secondary closure	Retained large metallic foreign body left upper lobe, with associated intrapulmonary abscess. Bronchopleural fistulae	Penicillin protection 48 hours. Thoracotomy with decortication. Débridement closure of abscess. Local penicillin. Penicillin 10 days postoperative	Complete primary cure. No complications.
23.	SFW perf. multiple right transdiaphragmatic	34 days	47 days	Liquid	*Hemolytic Staphylococcus aureus*	Early thoracoabdominal E. H. Rib resection drainage for empyema	Multiple bronchopleural fistulae	Penicillin protection 17 days. Thoracotomy with decortication. Local penicillin. Penicillin 14 days postoperative	Basal empyema requiring rib resection drainage. Persistent anterior empyema cavity, may require future operative procedure.
24.	SFW pen. left	50 days	61 days	Liquid	*Anaerobic Streptococcus. Pseudomonas*	Early thoracotomy E. H. Rib resection drainage for empyema	Multiple bronchopleural fistulae	Penicillin protection 11 days. Thoracotomy with decortication. Local penicillin. Penicillin 16 days postoperative	Recurrent basal empyema requiring secondary rib resection drainage.
25.	Multiple perf. SFW severe	18 days	28 days	Clotted	*Proteolytic Clostridia Sporogones*	Rib resection drainage for empyema	Multiple bronchopleural fistulae. Multiple small intrapulmonary abscesses, with long connecting missile tracts	Penicillin protection 72 hours. Thoracotomy with decortication. Local penicillin. Penicillin 16 days postoperative	Basal empyema. Recurrence of bronchopleural fistulae. Bronchopneumonia contralateral side. Death after 8 weeks.

Explanation of abbreviations.
SFW — shell fragment wound.
GSW — gun shot wound.
pen. — penetrating.
perf. — perforating.
E. H.— evacuation hospital.

current basal empyema following decortication, one (Case 18) occurred in a patient who had a fracture of the femur treated by traction throughout his postoperative thoracotomy course. The immobility of the patient thus engendered, we feel to have been contributory to the failure to obtain a primary cure in this case. It is to be stressed here that whenever long bone fractures occur in association with significant thoracic trauma the fracture should if possible be handled in such a fashion as to render the patient maneuverable. In our experience, a thoracic injury that is immobilized by skeletal traction for the treatment of a long bone fracture is very likely to do badly so far as the chest lesion is concerned. The second case with recurrent empyema in this group (Case 13) presents no reason, so far as we can see, for having failed to achieve a primary cure.

It is significant that of the six cases of recurrent empyema, four occurred in the second group; *viz.*, those with significant associated pulmonary pathology. These associated lesions were serious and complicated. A review of these lesions as summarized in Table I is interesting. Three cases of pulmonary abscess were encountered. All three were débrided and the remaining defects closed. In two of these (Cases 7 and 22), complete healing of the abscess with primary cure of the empyema was obtained. The third abscess (Case 21), much larger, reopened, and gave rise to a recurrent empyema. Despite this, we feel that these cases justify the validity of the method, and that they are striking evidences of the marked extension of surgical therapy that penicillin protection permits of in the eradication of sepsis.

In only one of the cases (Case 23) is there any likelihood that subsequent additional surgery will be necessary to effect a cure.

The consistently uneventful convalescent period and lack of any evidence of dissemination of infection is best appreciated perhaps by a study of the temperature and pulse records of the patients. For this purpose Figures 4, 5, and 6 are included. Figure 4 is the temperature and pulse chart of Case 8, Figure 5 is the temperature and pulse record of Case 20, and Figure 6 is that of Case 22. The absence of any marked reaction to operation is striking and was characteristic of the entire series. Even in the case that eventuated fatally (Case 25) the early postoperative course was favorable. Defervescence has been prompt in all cases. Where the empyema has recurred the temperature reaction was minimal.

The prompt and complete degree of pulmonary reëxpansion obtained in the 19 cases of primary cure is illustrated by the series of photographs of pre- and postoperative roentgenograms, which are included (Figs. 7, 8, 9, 10, 11, and 12). All of these were made within three weeks from the time of operation. They clearly demonstrate the early return to normal and the lack of any evidence of residual disease.

Not the least gratifying and impressive aspect of this series was the short time-interval necessary to effect a cure. Beside the weeks of daily dressings and tube changes required by the other methods these cases present a very

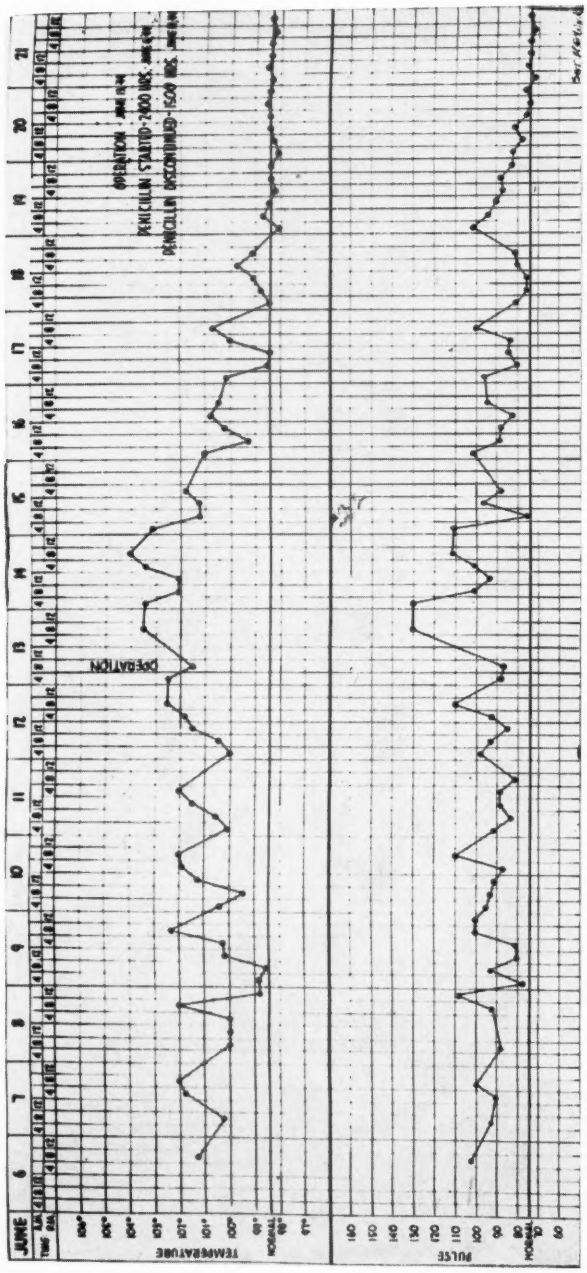

FIG. 4.—Temperature chart of Case 8 showing reaction to operation and prompt defervescence following decortication.

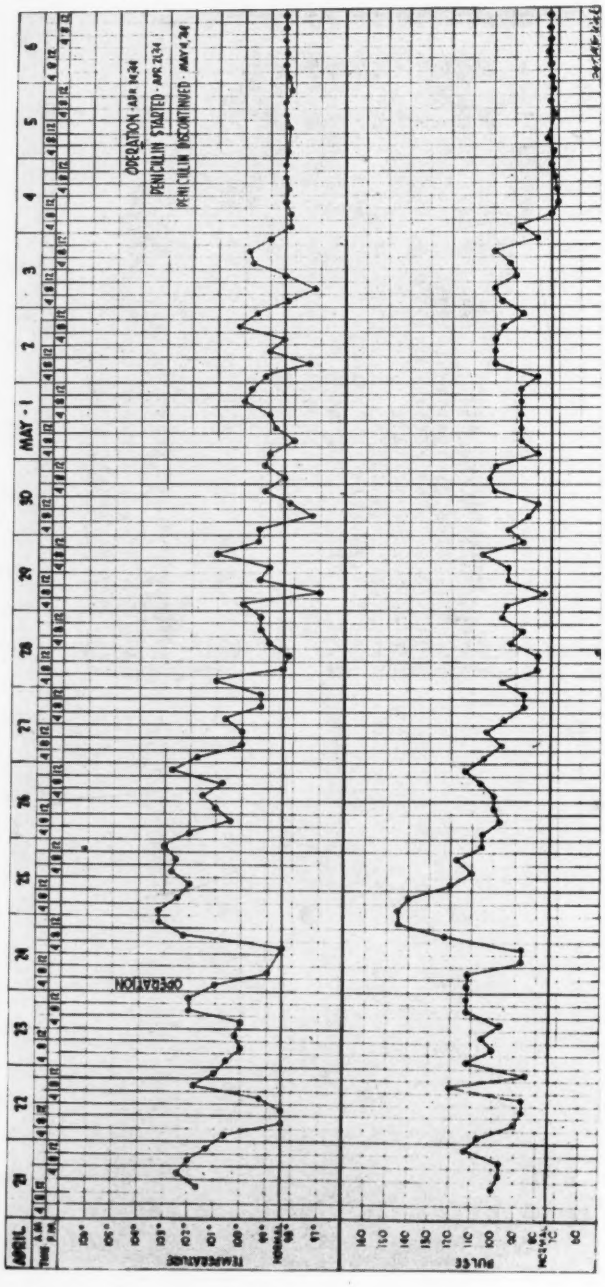

FIG. 5.—Temperature and pulse chart of Case 20 showing minimal reaction to operation and prompt return of pulse and temperature to normal.

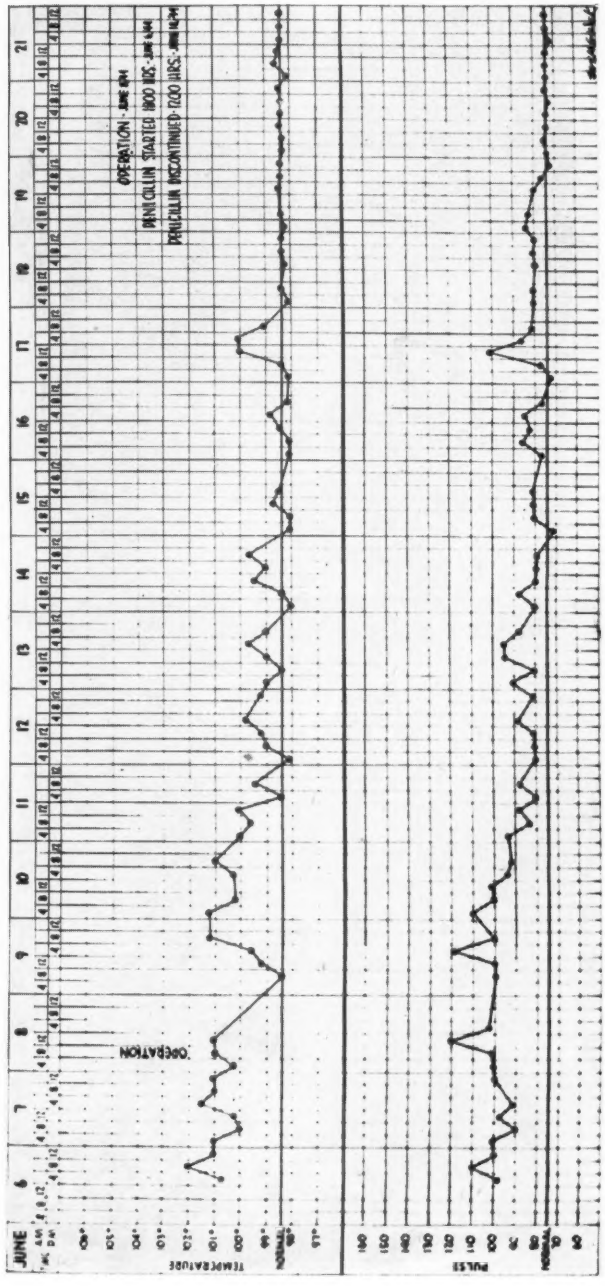

FIG. 6.—Temperature and pulse chart of Case 22 showing absence of any untoward reaction to operation and prompt lysis to normal temperature. The temperature spike on June 17 followed removal of the posterior intercostal tube.

FIG. 7

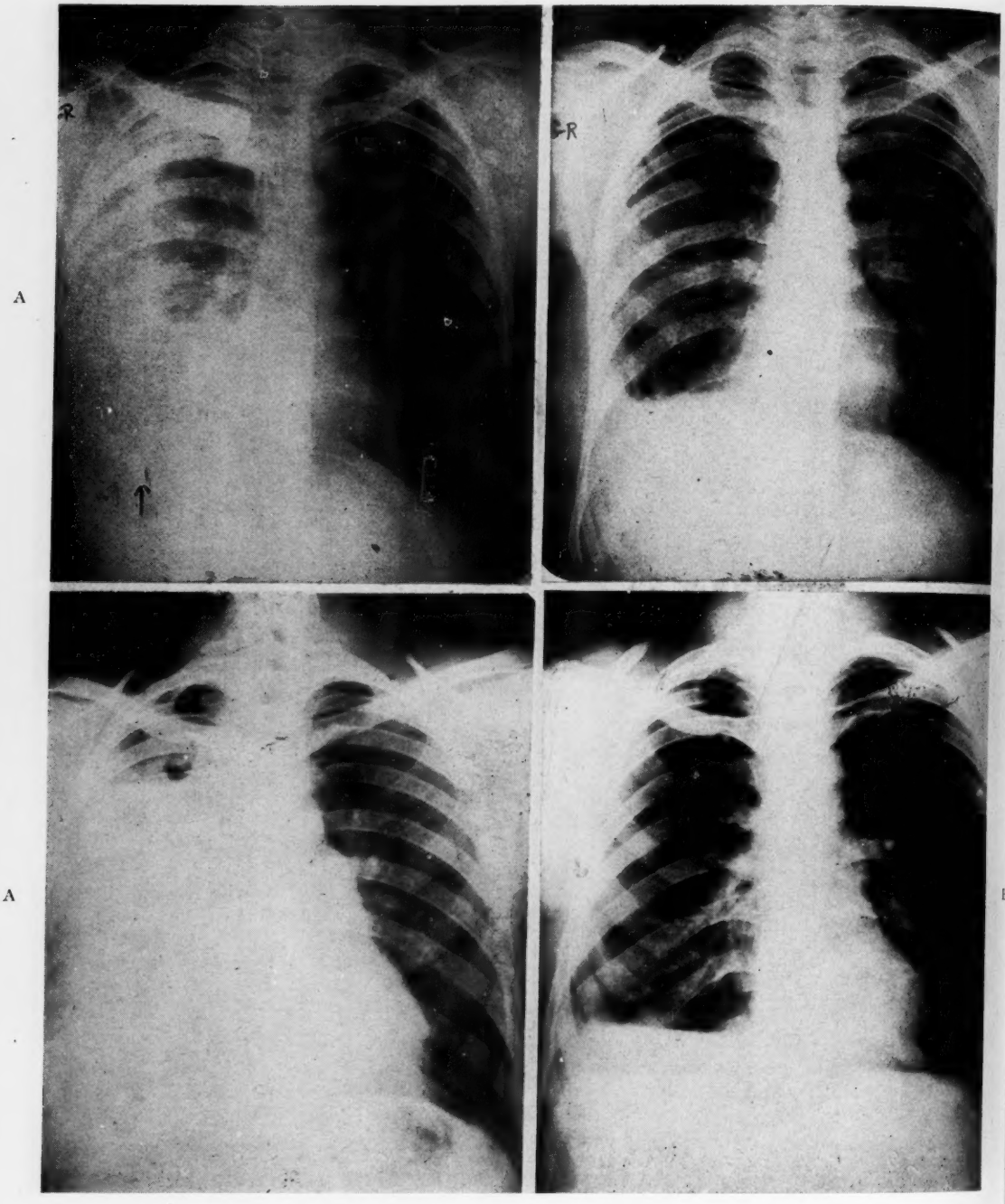

FIG. 8

FIG. 7.—Case 6: A. Roentgenogram of chest at the time of operation showing empyema of right pleural cavity with intrapleural foreign body.
B. Roentgenogram of chest three weeks after thoracotomy with decortication.
FIG. 8.—Case 8: A. Roentgenogram of chest showing organizing hemothorax, with empyema of right pleural cavity.
B. Roentgenogram showing chest two weeks after thoracotomy with decortication.

FIG. 9

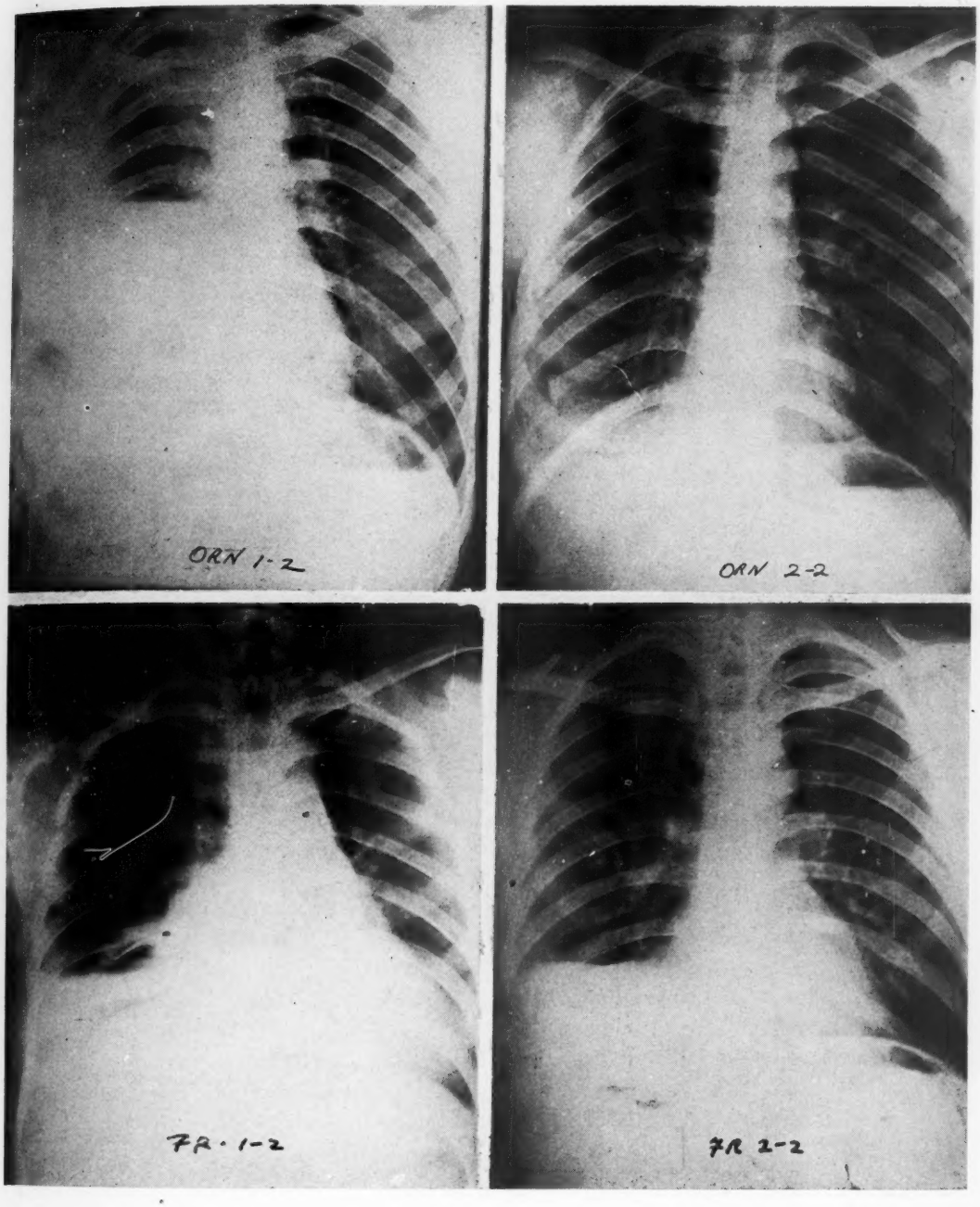

FIG. 10

FIG. 9.—Case 10: A. Roentgenogram of chest showing empyema of right chest. Patient was operated upon at this stage.

B. Roentgenogram of chest two weeks after thoracotomy with decortication.

FIG. 10.—Case 17: A. Roentgenogram of chest showing empyema of right chest, with complete collapse of right lung.

B. Roentgenogram of chest three weeks after thoracotomy with decortication showing complete expansion and no empyema.

FIG. 11

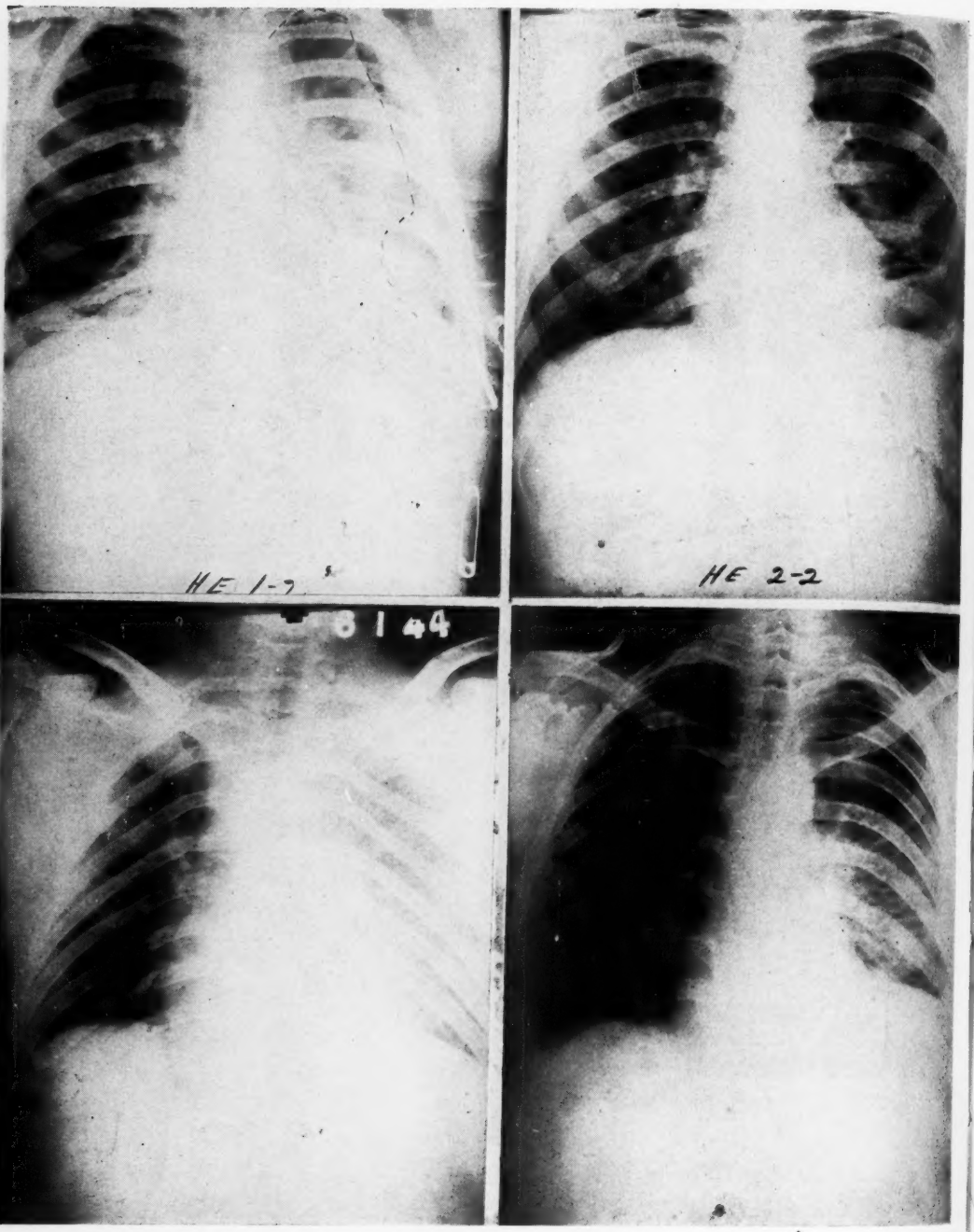

FIG. 12

FIG. 11.—Case 20: A. Roentgenogram of chest showing left-sided empyema of six weeks duration. B. Roentgenogram of chest one month after thoracotomy with decortication showing complete cure.
FIG. 12.—Case 22: A. Roentgenogram of chest showing left-sided empyema thoracis. This patient was found at operation to have a definite abscess of the upper lobe associated with the intrapulmonary foreign body.
B. Roentgenogram three weeks postoperatively.

striking and satisfactory contrast. With solid, cleanly healed wounds and totally expanded lungs the primary cures are very shortly back on the road to complete convalescence. Figure 13 is a striking photograph of a group of five of the 19 primary cures and illustrates their physical well-being within two to three weeks after thoracotomy with decortication.

The disposition of the 24 surviving patients in this series was as follows: Nine were returned to duty in this Theater. Of these, two were sent to full duty, and seven to limited duty, with the recommendation that their duty

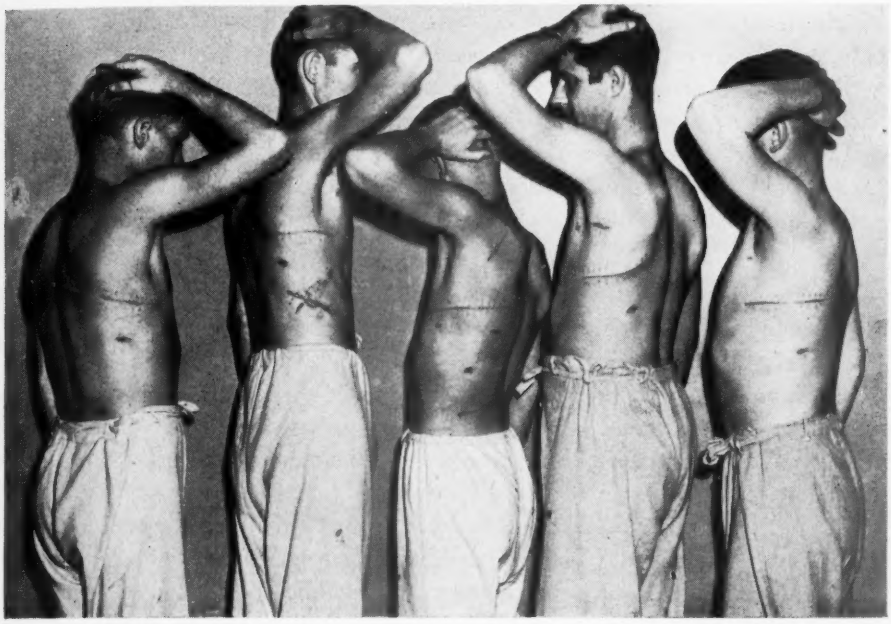

FIG. 13.—Photograph showing five of the cases presented in the text. These cases all had definite empyema, and are shown two and three weeks after thoracotomy with decortication. All in this group returned to duty.

status be reviewed within 90 days. The remaining 15 were sent to the Zone of the Interior. This group included the five recurrent empyemas who survived. Two of the remaining ten were sent to the Zone of the Interior because of concomitant lesions. The remaining eight, though cured of their empyema, had not progressed quite to the point, so far as vital capacity and general physical condition was concerned, where it was felt advisable to return them to duty in this Theater.

From our experience we are convinced that a proper alherence to the principles we have discussed should result in a 90 to 95 per cent primary cure-rate in those cases having only pleural involvement. In those having significant associated pulmonary damage the incidence of recurrent empyema will be higher. However, the process will be greatly shortened and the chances of a chronic empyema will be markedly minimized. Those requiring subsequent mutilating obliterative operations should be very few indeed.

To those who would hold a brief for a more conservative approach to the therapy of posttraumatic empyema we emphasize the not insignificant mortality of the adequately drained empyema that "is doing well." Even the best managed case is unpredictable and the road from drainage to complete cure is beset with significant dangers. It requires but one metastatic brain abscess to emphasize this. Chronicity may eventuate at any stage. At best, the time elapsing before a cure is obtained is a matter of weeks. Uncertainty of outcome is inevitable so long as any vestige of an empyema cavity exists.

From a study of the 25 cases in this series we believe that early thoracotomy, with decortication, under penicillin protection is the method of choice in the therapy of total posttraumatic empyema. We feel that we can now approach the problem of posttraumatic empyema more rationally and more optimistically, certain of achieving a high percentage of primary cures, with early, complete functional restoration and a greatly lowered chronicity rate, with a minimum of risk to the patient.

SUMMARY AND CONCLUSIONS

1. The significance of the problem of posttraumatic empyema thoracis is presented and the basic pathology of the problem, which has been studied intensively, is discussed.

2. A concept of therapy has been rationalized on a basis of that pathology, and a plan of treatment is presented which is based on that concept. The treatment recommended consists of early pulmonary decortication under penicillin protection.

3. A series of 25 cases treated by this method are reviewed. Of this group, 19 (76 per cent) obtained a primary cure. Six developed recurrent basal empyemas. In only one is there any likelihood of a subsequent obliterative operative procedure being necessary to bring about a cure. The one death in the group occurred eight weeks after operation of continued sepsis, and is not classed as an operative death.

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Care of Office of the Surgeon General
Washington 25, D. C.

TRAUMATIC RIGHT DIAPHRAGMATIC HERNIA

CASE WITH DELAYED HERNIATION OF THE LIVER AND GALLBLADDER

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TRAUMATIC HERNIA rarely occurs through the right hemidiaphragm. In one large series its incidence is given as 5 per cent. In 857 cases of traumatic diaphragmatic hernia, the liver was found in the chest in but 14 cases.[1] The following case is reported because of several notable features: The herniation of the liver and gallbladder occurred through a rent in the right diaphragm late after the injury; the symptoms occasioned by the displaced organs were moderate, and the exact status of the herniated structures could be inferred from the roentgenologic examinations.

Case Report.—A 25-year-old bombardier received crushing injuries to the right lower chest, the right thigh, and the left forearm when his plane crashed in a faulty take-off. Upon admission to a Station Hospital he complained of pain in the right lower chest and right upper abdomen, stating that he felt as though all the wind were knocked out of his right lung. Roentgenograms demonstrated comminuted fractures of the middle third of the left radius and ulna, and an oblique fracture of the middle third of the right femur. A roentgenogram of the chest showed elevation of the right dome of the diaphragm to the level of the eighth intercostal space posteriorly; the heart and lungs were otherwise normal. He was observed for signs of a ruptured viscus or internal hemorrhage, but these did not materialize. Adhesive skin traction was applied to the right leg and the left forearm was placed in a plaster encasement. A course of sulfadiazine therapy was started immediately. For five or six days he complained of constant right chest pain, which gradually diminished. The pulse remained at about 110, and the respirations at about 25 per minute. He developed a slight productive cough for several days, but at no time did he expectorate blood. Ten days after admission he was transferred to this General Hospital.

Physical Examination.—The patient was a young male whose left forearm was in a plaster encasement, and whose right leg was in traction in a Thomas caliper splint. His temperature, pulse and respiratory were normal. Healing recent lacerations of the left elbow and right thigh were present. Although he complained of slight discomfort in the right lower chest on deep inspiration, the examination of the chest failed to show any abnormalities. The blood, urine and serologic tests were also normal. A roentgenogram of the chest showed slight elevation of the right diaphragm (Fig. 1).

After preliminary blood and plasma infusions an open reduction of the left forearm was undertaken. Skeletal traction was applied to the right femur. These procedures were performed under inhaled nitrous oxide-ether anesthesia, with intravenous sodium pentothal induction. No respiratory difficulty was encountered during the 100 minutes of anesthesia. The patient's convalescence was uneventful.

About three months later the patient began to complain of pain in the right lower chest, which was made worse by the ingestion of food. Peristaltic gurgles were heard high in the right axilla. A roentgenogram of the chest showed a large homogeneous density occupying the lower half of the right hemithorax (Fig. 2). Fluoroscopy in the erect and recumbent positions failed to show any change in the shape of the dense shadow, or any evidence of movement of the shadow with respiration. The films of

the abdomen and the chest showed gas-filled bowel, which was thought to be colon, high in the most lateral portion of the lower right axilla. The usually sharp lower margin of the liver could not be seen (Fig. 2). Examination of the colon by means of a barium enema verified the fact that the hepatic flexure was unusually high in position, and, again, failed to visualize the lower margin of the liver. It was postulated, therefore, that the liver had migrated into the lower portion of the right chest. In order to confirm this, cholecystography was done. This examination showed a markedly elongated narrow gallbladder, the fundus of which was directed upwards towards the right axilla (Fig. 3). Concentration and emptying after a fatty meal were good.

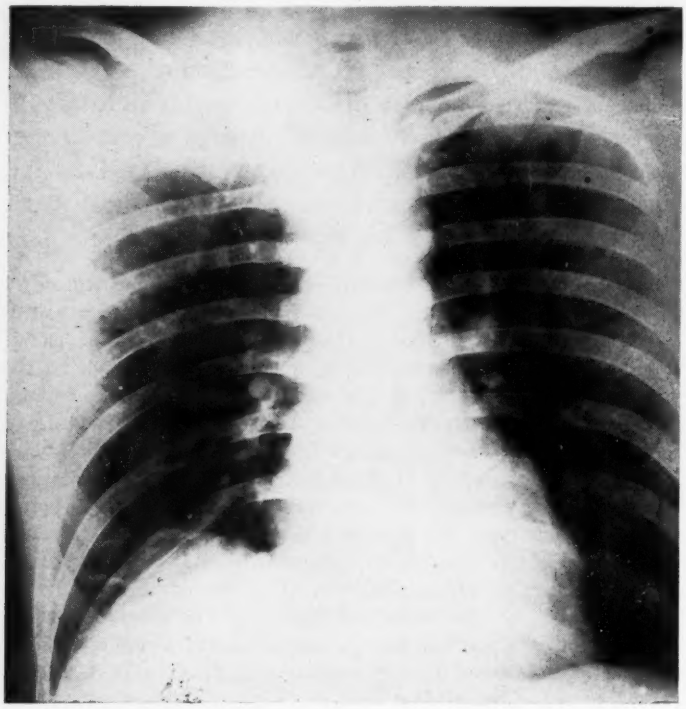

FIG. 1.—The right hemidiaphragm is somewhat elevated. This examination was made under bedside conditions on admission of the patient to this hospital.

At this time it appeared that the patient would need an open reduction of the femur, as well as a repair of the diaphragmatic hernia. It was decided to place the right leg in a long leg encasement, and to repair the hernia first. Vitamins and multiple blood and plasma infusions were given preoperatively. He was allowed up in a wheel chair, with improvement in appetite, bowel function, and general attitude.

A month after the onset of the new group of symptoms the right diaphragmatic hernia was repaired. With the patient lying on his left side, a long skin incision was made on the right side between the seventh and eighth ribs from the costochondral junction to the tubercle of the ribs. The seventh, eighth and ninth ribs were exposed in the posterior axillary line and two drill holes made in each rib about 2 cm. apart. These ribs were then cut transversely between the drill holes. The pleural cavity was opened between the seventh and eighth ribs and a self-retaining retractor inserted. This exposed the gallbladder and the under surface of the liver. This latter organ was lying in the thoracic cavity, completely upside down. The right hemidiaphragm was contracted and shrunken posteriorly and medially, and lay under the rotated liver. The

liver had turned 180° on its transverse axis, so that the inferior surface was lying superiorly. The gallbladder was stretched into a long thin tube-like structure which extended from the posterior superior portion of the chest down into the abdomen. The left lobe of the liver was displaced to the right of the round ligament and had rotated about 120°. The portal vein and the gastrohepatic omentum were stretched tensely, but not enough to impede the flow of blood or bile. A moderate amount of omentum and the tip of the hepatic flexure of the colon were above the diaphragm posteriorly.

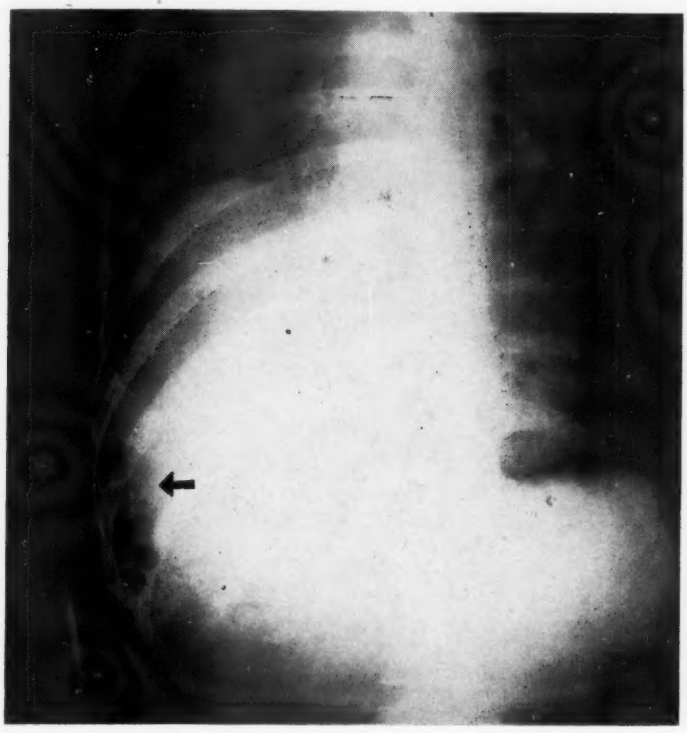

FIG. 2.—A homogeneous shadow is seen in the lower half of the right side of the chest. Its upper border did not change with respiration or with change in posture. The heart is displaced to the right. Gas in the bowel, presumably colon, is present high in the abdomen (arrow). The usual lower margin of the liver cannot be seen. The patient is in a spica encasement.

There was a small amount of serous fluid posteriorly in the chest cavity. There were no adhesions, nor was there any evidence of previous hemorrhage or fracture of the liver. It was seen that the diaphragm was torn completely and cleanly from its attachment beginning near the sternum and extending entirely around the chest wall to the posterior axillary line on the right side. The free edge of the diaphragm and the site of its normal attachment were smooth and nicely healed. The peritoneal margin along the anterior abdominothoracic junction was clearly demarcated.

The liver was rotated into normal position with slight difficulty and simultaneously the omentum and colon were reduced into the abdominal cavity. The diaphragm was then pulled forward and laterally with ease, and its edges sutured to its previous attachments by multiple interrupted mattress sutures of No. 3 black silk. Interrupted sutures of No. 5 black silk were also used about 3 cm. apart. This effected an anatomic reposition of the diaphragm. The phrenic nerve was exposed above the diaphragm and crushed, resulting in an immediate cessation of diaphragmatic activity. During the

operation there was a marked mediastinal shift to the left, but respirations were controlled satisfactorily with positive pressure anesthesia. Stainless steel wire was then put through the holes in the ribs which had been severed, and anatomic position was restored. The pleura, except for the most mesial portion, was closed with interrupted sutures of "o" catgut chromic. The rest of the wound was closed in layers using the same suture material. Two loops of No. 5 chromic catgut were placed about the seventh and eighth ribs and tied to relieve tension on the soft tissue sutures. The mesial aspect of the wound was left open and interrupted sutures placed but not tied in all the layers. All fluid and blood was removed from the chest by suction, and the lung was expanded by positive pressure. The wound was then quickly closed by tying

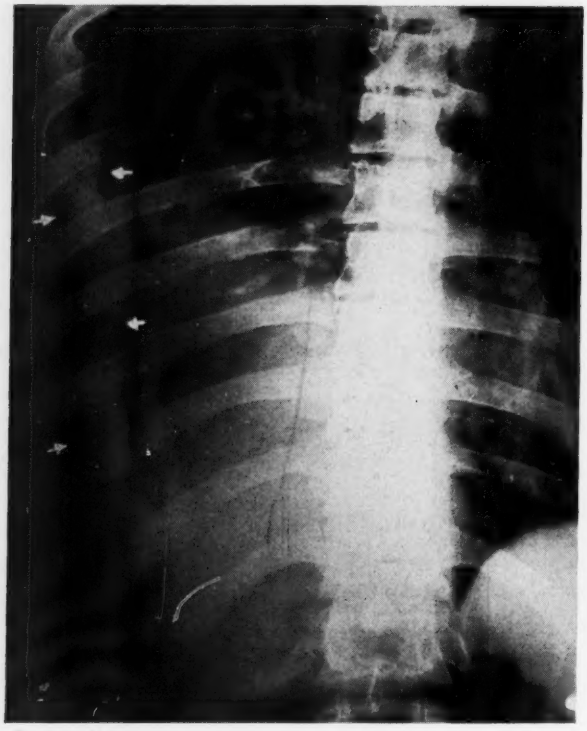

FIG. 3.—Cholecystography shows a long tube-like gallbladder stretching to the level of the 5th rib posteriorly. This shadow changed in size after a fatty meal.

the previously placed sutures, thereby obtaining an air-tight closure. The skin was closed without drainage, using interrupted black silk sutures.

He was immediately started on penicillin systemically, 25,000 units every two hours. This was continued until the ninth postoperative day. His course was uneventful, the temperature, pulse and respiration becoming normal on the sixth day. On the third day postoperatively, 800 cc. of blood-tinged fluid were removed from the right pleural cavity by thoracentesis. The operative wound healed *per primam*. Three 500 cc. transfusions of blood were given during the first ten postoperative days. The patient had no further trouble with his chest.

A reexamination of the gallbladder after the operation showed it to concentrate very well. It had resumed its normal position and shape. The liver shadow was also seen to be in normal position (Fig. 4).

DISCUSSION.—It is remarkable that the rotation of the liver did not produce obstruction of the portal or biliary system. It seems evident that the extensive diaphragmatic separation existed from the day of the accident, yet respiratory embarrassment and mediastinal shift did not occur. The liver itself probably plugged the gap and acted as part of the thoracico-abdominal septum. As time passed, the stretching of the attachments of the liver allowed it to prolapse into the chest. The contraction of the dia-

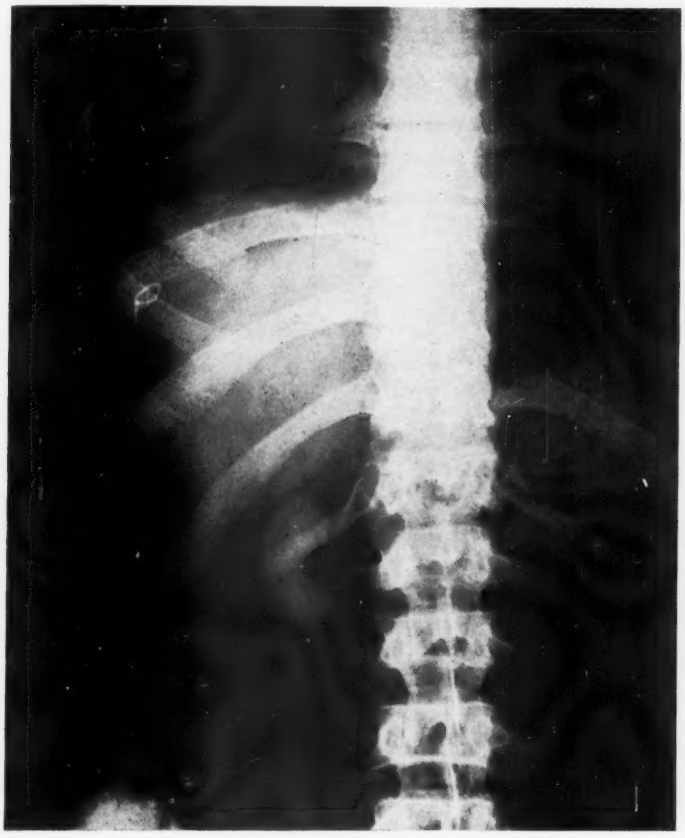

FIG. 4.—Postoperative cholecystography shows a gallbladder normal in size, shape, and position. The lower margin of the liver and the hepatic flexure of the colon, both now in normal position, can be seen.

phragm posteriorly and medially, exerting tension on the intact triangular ligament was probably a sufficient factor in initiating rotation of the liver. The constant action of the left hemidiaphragm transmitted through the abdominal contents completed the rotation of the liver and its upward displacement. Colon and omentum followed but due to the lack of inflammation these organs probably slid in and out of the chest with change in body position or physiologic activity. The absence of inflammatory reaction, of signs of previous hemorrhage, and the glistening smoothness of the peritoneal and pleural surfaces are unusual.

SUMMARY

A case is described in which the right hemidiaphragm was detached through approximately 70 per cent of its costal origin. About four months after the original injury the liver herniated into the chest, rotating 180° on its long axis as it did so. Roentgenographic studies, particularly those of the gallbladder, suggested the disordered anatomy which was found at the operation. Restoration of the viscera to normal positions and repair of the hernia were accomplished.

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THE VALUE OF ANTITOXIN IN THE PREVENTION AND TREATMENT OF MALIGNANT EDEMA AND GAS GANGRENE*

A REVIEW OF OBSERVATIONS

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THE HISTORY of malignant edema and gas gangrene may be divided into two periods, first, the prebacteriologic or purely clinical era, from 1607, when Hildanus first recorded a case[1], to 1877, when Pasteur and Joubert[2] discovered the *"vibrion septique,"* and, second, the bacteriologic era, from 1877 to the present.

Anyone interested in the prebacteriologic era of these anaerobic infections will find the work of Kellett[1], Kirkland[3], Martin de Bazas[4], Maissoneuve[5], Chaissaignac[6], Macleod[7], Pirogoff[8], and Trifaud[9], thoroughly worth while, but the present discussion relates exclusively to the problems of serum therapy. In order to appreciate this it is necessary briefly to discuss the complicated etiology of these diseases.

Malignant edema and gas gangrene are almost always the result of severe trauma coupled with excessive contamination with fecal micro-organisms present in the soil, upon clothing or other foreign objects carried deeply into the wounded tissues which are not removed promptly by surgery. Crushing injuries involving compound fractures of the limbs, gunshot wounds, bombing injuries, traumatic abortion, hypodermic injections and automobile and train accidents are the principal predisposing causes. Superficial injuries to the skin such as abrasions without crushing and even extensive burns rarely result in anaerobic infections. There are, thus, three main factors: (1) injury of a certain kind; (2) gross contamination with certain pathogenic bacteria; and (3) lack of adequate and prompt surgery. It is only when all three of these factors operate conjointly that malignant edema or gas gangrene result.

SURGERY

Surgeons have recognized for many decades that compound fractures could be successfully treated by adherence to the principles of prompt and adequate débridement, immediate fixation, as for example, in plaster of paris and provision for free drainage. In 1884, when the rôle of bacterial infection was only vaguely appreciated, Dennis[10] recorded 144 cases without a single death from septic infection and 100 cases without a death from any cause.

More recently, Böhler[11], after an amazing experience with 20,000 open traumatic wounds, including 253 compound fractures of the long bones and

*This work was done under a contract recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Columbia University, New York, N. Y.

Dr. Frank L. Meleney of the Subcommittee on Surgical Infections was the Responsible Investigator.

more than 100 open tears of large joints, treated surgically, but without serum, lost only one patient from gas gangrene, while Trueta[12, 13], had only one case of gas gangrene in over 1,000 compound fractures during the Spanish Revolution, relying mainly upon prompt, meticulous surgery, with immediate fixation in plaster.

These observations prove beyond question that if early débridement and adequate surgery are practiced, anaerobic infections are extremely rare. The real value of serotherapy in the treatment of wounds lies in the fact that it is so simple that it can often be administered very soon after a wound is received, making delayed surgery possible and effective, saving lives and limbs that would otherwise be lost. Essentially, the value of serotherapy is prophylactic, and to a much lesser degree, therapeutic.

Bacteriologically, the early conceptions of monospecific etiology based upon the work of Pasteur and Joubert[2] on *"vibrion septique,"* and of Fraenkel[14] upon *Bacillus phlegmonis emphysematosae,* later shown[15, 16] to be identical with the *Bacillus aerogenes capsulatus* of Welch and Nuttall[17] and now properly called *Bacillus perfringens*[18], gradually gave way in the early part of the century to the present conception that anaerobic wound infections, including malignant edema, gas gangrene and tetanus, are rarely pure infections but generally polymicrobic mixed infections in which two, three, or many species of bacteria, aerobic and anaerobic, pathogenic and saprophytic, putrefactive and fermentative, may participate. This change in point of view has been most clearly enunciated by Weinberg and Sequin[19].* At the close of World War I the principal primary cause of anaerobic wound infections other than tetanus were regarded as *Bacillus perfringens, Bacillus septicus, Bacillus novyi* and *Bacillus histolyticus.* To these has been added *Bacillus sordellii*[20, 21, 22, 23]. These five anaerobic bacilli are the principal specific causes of anaerobic wound infections other than tetanus, but their clinical manifestations are greatly modified by the various combinations in which they occur among themselves and with numerous aerobic and other anaerobic bacteria.*

SEROTHERAPY

The first attempt at serotherapy in this connection was made by LeClainche[24], in 1898, when he prepared an antimicrobic serum in an ass which would protect guinea-pigs and rabbits against several fatal doses of a culture of *"vibrion septique,"* and would sometimes save their lives when given an hour after the culture was injected. In 1901, LeClainche and Morel[25] showed

*In this review the names used by various authors will be cited, but in case of doubt as to identity the following list of synonyms may be consulted:

Bacillus septicus, vibrion septique, Clostridium septicum, Clostridium septique. Bacillus tetani. Clostridium tetani. Bacillus perfringens, Bacillus aerogenes capsulatus, Bacillus phlegmonis emphysematosae, Bacillus welchii, Clostridium perfringens, Clostridium welchii, Fraenkel's gas bacillus. Bacillus novyi, Bacillus oedematis maligni II, Bacillus oedematiens, Clostridium novyi, Clostridium oedematiens. Bacillus histolyticus, Clostridium histolyticum. Bacillus sordellii, Bacillus oedematis sporogenes, Clostridium oedematoides, Clostridium sordellii.

that animals receiving a "serum-virus" mixture had no permanent immunity to *"vibrion septique."* No attempt seems to have been made immediately to apply such a serum in human cases of anaerobic cellulitis, which, at that time, was generally believed in France to be caused by the *"vibrion septique."*

Prior to World War I *B. perfringens* was vaguely supposed in France to be a possible cause of acute articular rheumatism, and a serum was prepared in horses by Thiroloix and Rosenthal[26] which in small quantities would protect guinea-pigs if given before, or simultaneously with, normally fatal doses of culture, and would even save them if given within three hours afterward in larger doses. Rosenthal[27] used this serum in proving the identity of *B. perfringens* with Achalime's anaerobic bacillus of rheumatism and later suggested its therapeutic application[28], but we have no record of any observations in this direction. Neither is there any indication in the literature that the relation of *B. perfringens* to gas gangrene was appreciated at all in France prior to the outbreak of World War I.

In America, on the contrary, Welch and Nuttal's "gas bacillus" was widely accepted as the essential cause of gas gangrene, but the common method of treatment, as summarized by Cramp[29], in 1912, was one of conservative surgery under spinal anesthesia, amputation when indicated, leaving wounds wide open, frequent inspections and irrigations with H_2O_2.

Shortly after World War I began it was realized that gas gangrene would be a major problem, and Weinberg, who was assigned to the study of the English wounded, soon recognized the importance of *B. perfringens* and of mixed infections as well as the infrequency of *"vibrion septique."* Almost at once he began to experiment first with vaccines[30], and soon after with antiserums against *B. perfringens*[31]. In 1915, after studying about 80 cases, a soldier treated with antiperfringens serum died from a *"vibrion septique"* septicemia. Weinberg's determination to prepare a bivalent serum against gas gangrene dates from this incident[32].

About the same time Raphael and Frasey[33] reported a successful antitoxin against *"vibrion septique"* and, in 1916, a bivalent serum was made by Le Clainche and Valee[34] against *B. perfringens* and *"vibrion septique."*

In 1916, Weinberg[35, 36] was still talking about the use of his "omnivalent iodized autovaccine" but he was also hopeful of antitoxic and antimicrobic serums against *B. perfringens, "vibrion septique"* and *B. oedematiens.* In 1917, Weinberg and Sequin[37] produced a successful serum against *"vibrion septique,"* and a little later *B. perfringens* and *B. oedematiens* as well[38]. With this trivalent serum they reported 19 cures in 30 cases, whereas, in a group of 66 cases treated without serum there were 35 deaths. Further tests were made of Weinberg and Sequin's serum by Vaucher[39], and of both this and LeClainche and Valee's serum by Ivens[40] on larger numbers of cases, with encouraging but inconclusive results. Individual cases, such as that of Delbet[41], often responded brilliantly, and LeClainche[42] reported 31 out of 40 cases as cured by his serum. Duval and Vaucher[43] injected 50 men from 5 to 18 hours after they were wounded, with either *B. perfringens* and *B. oede-*

matiens antiserum, or *B. perfringens* and *"vibrion septique"* antiserum or a mixture of these, all prepared by Weinberg and Sequin. Twenty-five of these patients died within 24 hours on account of their severe wounds, but without gas gangrene; 24 lived under observation for periods of 8 to 28 days without developing gas gangrene. These observers concluded that (1) prompt serotherapy was fully justified; (2) it could not be regarded as a substitute but as an important adjuvant of surgery; and (3) it had definite therapeutic value also. Subsequently these authors[44] reported the survival of all but two of 74 severely wounded men given these serums within 48 hours, without gas gangrene and without amputations. As a result of this report the Societe de Chirurgie de Paris voted to adopt the use of these serums in all wounded men likely to develop gas gangrene. A little later, Duval and Vaucher[45] reported on the preventive inoculation of 449 wounded men of whom 55 died from trauma without gas gangrene. Thirteen men should not have been injected. There were only 18 cases (4.7 per cent) of gas gangrene among 381 which might have occurred. Of these 18 cases, 10 died while 8 recovered after amputation of damaged limbs. In control groups not treated the number of cases ran 15 to 18 per cent.

Ivens[46] reported essentially similar favorable observations on 376 cases of wounds of which 236 had fractures, treated either with Weinberg's serum or that of LeClainche and Vallee.

Mairess and Regnier[47] treated 297 wounded men with evidence of anaerobic bacteria in their wounds, with serums from the Institute Pasteur. Of these, 25 developed gangrene and required secondary serotherapy. Only five died of gas gangrene. The clinical impressions were regarded as very favorable.

Vincent and Stodel[48] observed that under the influence of serum the general and local symptoms mended rapidly, the pulse and fever were lowered, the urinary secretion increased, and the wounded men's spirits revived. Of 81 treated, 12 died but only 8 died from gas gangrene.

It should be noted here that all of the above observers insisted upon the prompt use of the serum in fairly large amounts, which could not be clearly defined because of lack of any real method of standardization. None decried the absolute necessity of surgery and none regarded serotherapy as any substitute for surgery.

In Germany, a "Gasbrandserum gegen der Fraenkel'schen Gasbacillus" was prepared by Fraenkel and Zeissler[47] of which doses of 0.2 cc. intraperitoneally would protect guinea-pigs against many fatal doses of culture given intramuscularly four hours later, while polyvalent antigas gangrene serums of somewhat uncertain composition were prepared by Aschoff[50], and by Kolle, Sachs and George[51], which prevented experimental infections in guinea-pigs, and saved about 44 per cent of wounded soldiers threatened with gas gangrene.

It seems clear that the trivalent serum made by Weinberg and Sequin, in 1916 and 1917, contained antitoxins for *B. oedematiens* (*B. novyi*) and *"vibrion septique"* (*B. septicus*), but there was no evidence then for anything but

antibacterial antibodies against *B. perfringens*. Bull and Pritchett[52], in 1917, first demonstrated the production of a true though weak, exotoxin by *"Bacillus welchii"* (*B. perfringens*), and prepared an antitoxin in rabbits which would neutralize its action in pigeons. Later, Bull[53] prepared a serum from a horse which arrested and controlled established infections and conferred passive immunity durable for two weeks in guinea-pigs. A few cases of gas gangrene in man were treated "in which the efficiency of the antitoxin was unmistakable[54]."

Wilson[55], analysing 76 cases of gas gangrene occurring at a Base Hospital, felt after the war that the case for serum treatment had not been proven clinically, but Van Beuren[56] summarizing the best practice at the close of the war, and giving full data on numbers of wounded and incidence of gas gangrene in World War I, was favorable to the use of serum as an important adjunct to surgery. Equally favorable were the reports of Haniquet[57], Sacquepee[58] and Vincent[59, 60]. Sacquepee pointed out that among 191 cases of gangrene treated with trivalent serum there were 160 cures and only 25 deaths, a mortality of about 13 per cent as compared with 75 per cent in untreated cases in the same region. Among 319 cases of severe wounds treated preventively only four cases of gas gangrene occurred.

One of the best reports on the treatment of gas gangrene, as it was known at the close of the war, was that of Ireland, *et al*[61]. This was also favorable to the use of gas gangrene serum.

USE OF GAS GANGRENE SERUM IN CIVIL PRACTICE

After the war it was several years before reports of the use of gas gangrene serum in civil injuries began to appear. Many of these reports are notable for their obvious lack of adequate bacteriologic data. In many instances there is nothing but the bare statement that "the gas bacillus" was demonstrated microscopically, and in some cases by culture. It is obvious that isolation in pure culture was rarely attempted, so that the problem of actual specific identity of either "the gas bacillus" and of the commonly associated other bacteria, both aerobic and anaerobic, is left untouched in these reports. Many are also reports of one or a few cases insusceptible even to the crudest statistical analysis; there is no point in citing these. Furthermore, the antibody content of the serums used was rarely described accurately; in fact the antibody content of commercial gas gangrene serums has been a subject of frequent discussion and change for many years, and serums made by different companies often differed not only qualitatively in the specific antibodies represented but quantitively as well. It is only in the last few years that official international standards have been available for the group of antitoxins represented in the polyvalent gas gangrene serum. The present writer[62] has only recently pointed out how unsatisfactorily and confusing the present standards are. The international units of antitoxin are so defined that no two represent the same protective power; the unit of *Bacillus sordellii* antitoxin is about 50 times as strong as that of *Bacillus perfringens* while that of *Bacillus novyi* is

about 100 times as strong. Under the circumstances, the data available in the literature do not seem suitable for comparative analysis but the over-all impression is favorable to the use of serum as an adjunct to surgery.

Among those whose experience with series of cases published during the decade 1927 to 1937 entitles them to express opinions favorable to the use of serum were Tenopyr[63], Weintrob and Messeloff[64], Boland[65], Larson and Pulford[66], Milch[67], King[68], Stone[69], Warthen[70], Ghormley[71], Eliot and Easton[72], Velicanov[73], Davis and Hanelin[74], Coller[75], Bates[76], Veal[77], and Eliason, Erb and Gilbert[78].

Illustrating a general point of view, in 1937, Bates reported 16 cases treated surgically without serum, of whom eight (50 per cent) died, while of 16 cases treated with serum as an adjunct to surgery, only three (18.7 per cent) died, and Veal recorded 13 deaths (48.1 per cent) out of 27 cases at Charity Hospital, in New Orleans, treated therapeutically but not prophylactically with serum, as against four (80 per cent) out of five without serum, two (7.4 per cent) out of 27 treated prophylactically, and ten (20.4 per cent) out of 49 treated both prophylactically and therapeutically. Eliason, Erb and Gilbert concluded that, "the value of serotherapy seems to be well established."

CHEMOTHERAPY

EXPERIMENTS UPON ANIMALS

Although many different antiseptics have been tried in the treatment of gangrenous wounds throughout the years, the first serious attempt to apply the principles of modern chemotherapy was made in 1937 when Domagk[79] stated that infections with *"C. welchii"* and *"C. septicum"* responded well to sulfanilamide compounds (uleron) with a substituted sulfamino group. However, Long and Bliss[80, 81] obtained poor results in mice inoculated with whole toxic cultures but with cultures centrifugalized and resuspended in glucose broth and inoculated intraperitoneally, which killed only 90 per cent of the controls, they were able to save from 44 to 100 per cent of the mice treated within an hour with 28 to 96 mg. of sulfanilamide, while none of those receiving 20 mg. survived. They interpreted this action of sulfanilamide as purely bacteriostatic.

Spray[82] found that the bacteriostatic action of prontosil-soluble, sulfanilamide and disulfanilamide increased in the order named against B. *tetani,* B. *novyi, B. septicus* and *B. histolyticus,* but that *B. welchii* and certain putrefactive anaerobes were scarcely affected *in vitro* under his experimental conditions.

Osgood and Powell[83], in 1938, showed that sulfanilamide did not inactivate the hemotoxins of *B. perfringens* or *"Cl. oedematis maligni"* (*B. septicus?*) *in vitro,* whereas, Carpenter and Barbour[84] found that sulfanilamide inactivated the toxins of *B. perfringens,* and other bacteria, *in vitro* and *in vivo* (mice) as well. These investigators were able with sulfanilamide to save 90 per cent of their mice injected intramuscularly with the toxin of *B. perfringens,* while only 12 per cent of the controls, not treated, survived.

Kendrick[85], on the contrary, was unable to demonstrate any significant therapeutic effect of neoprontosil, sulfanilamide or sulfapyridine in guinea-pigs inoculated with whole cultures of *B. perfringens* and found *B. perfringens* antitoxin much more effective.

In 1940, articles began to appear suggesting the possibility of synergic action when drugs were administered in conjunction with antitoxic serum. One of the first of these was that by Henderson and Gorer[86]. These investigators found that sulfapyridine was an efficient prophylactic and therapeutic agent against *vibrion septique* (*B. septicus*) inoculated intradermally into mice, but was unreliable when the mice were inoculated intramuscularly. *Cl. welchii,* Type-A, was susceptible to sulfapyridine only upon prophylactic inoculation of mice infected by the intradermal route. Sulfanilamide was less effective than sulfapyridine against both organisms, and there was no evidence that sulfapyridine given *per os* neutralized the toxin of either organism. It was found possible to control infections with *vibrion septique* by either antitoxin or antibacterial serum at a time when sulfapyridine was of little use, but the combined use of serum and sulfapyridine for both infections produced a noticeable synergic effect. These results were essentially confirmed by Stephenson and Ross[87] for *Cl. welchii,* Type-A and *"C. septique* (*sic*), but neither sulfanilamide nor sulfapyridine had any effect on *"Cl. oedematiens."* In Australia, Singer[88, 89] found that the use of antitoxic serums in mice infected with *C. welchii* or *C. septicum* made it possible to reduce greatly the effective dose of sulfanilamide but sulfanilamide was completely inactive against *C. oedematiens* and *Bacillus histolyticus.*

Gordon and McLeod[90] concluded from their experiments in mice and guinea-pigs, and other data, that the sulfonamide drugs were likely to have only a limited value in prophylaxis against gas gangrene, and that antiserums were much more effective in prophylaxis than drugs. Hawking[91] favored the combined use of sulfonamide drugs and antitoxin, and Reed and Orr[92] stated that "there is nothing to contraindicate the combination of these two forms of therapy."

OBSERVATIONS UPON PATIENTS

During the period of seven years in which the above experiments were recorded, many observations were also made upon accidentally infected wounds in human subjects. Lacking adequate controls, even bacteriologic analysis in many cases, and sometimes colored by wishful thinking, these are exceedingly difficult to evaluate. For example, Bohlman[93], who stated that the use of gas gangrene antitoxin had been disappointing in his hands, reported three cases as successfully treated with sulfanilamide. But each received 10,000 units of serum and there was apparently no examination to determine the species of bacteria present. Yet these cases have been frequently cited as proving the value of sulfanilamide in gas gangrene.

Kennedy[94], who condemned amputation as a means of saving life in gas gangrene, presented a case of gas gangrene in a boy following a shotgun wound treated with gas gangrene antitoxin, tetanus antitoxin, transfusion,

amputation (!) roentgen ray and sulfanilamide. There was no record of microscopic or cultural examination. Recovery was attributed to sulfanilamide!

Mellon, Gross and Cooper[95] described a case of "gas gangrene" following catheterization of an elderly man with chronic prostatitis and diabetes. *B. coli* was the only organism demonstrated. Treatment with gas gangrene serum gave only temporary relief, and the patient died after receiving large doses of sulfanilamide. This record is obviously unfair to both methods of treatment.

Of more value than any of these were Sadusk and Manahan's[96] two cases of postabortal septicemia, with positive cultures of *B. perfringens,* apparently saved by the oral administration of sulfanilamide. No serum was used in these cases.

Cruickshank[97], in an admirable discussion of the whole problem of infected wounds, and recognizing the doubt in the minds of some surgeons regarding the value of antitoxin, still favored the combination of sero- and chemotherapy. Ogilvie[98], likewise, favored the combined prophylaxis and therapy, and emphasized that "it is particularly important that serum should be given to those cases likely to develop gas gangrene, that is, men with lacerated wounds involving muscle who are unlikely to undergo débridement within the safe period." Lockwood[99], also, in a general discussion of sulfanilamide in surgical infections, concluded that "for the present it would seem that our chief reliance should be on prevention of gas gangrene by débridement of susceptible wounds and administration of prophylactic antitoxin." Warthen[100], on the other hand, in a summary of 71 cases of gas gangrene presented with incomplete bacteriologic data, was unable to find any single agent or drug specific in treatment, and stated that "perfringens antitoxin has been disappointing both prophylactically and therapeutically." Compare this with MacFarlane's[101] summary of 139 cases, in which "the fatality rate was significantly lower among patients who received antitoxin." MacFarlane emphasized "the necessity for the early and combined use of surgery, effective chemotherapy and antitoxin in the treatment of gas gangrene." The same general point of view was expressed by the British War Wound Committee[102], by MacLennan[103, 104] after an extensive experience covering four years in North Africa, and by Porter[105] in the South Pacific.

DISCUSSION AND SUMMARY

It seems clear from this study that whenever it is possible, as for example, in most sporadic civilian wounds, to undertake adequate surgery promptly, anaerobic infections may be prevented by this means alone, without recourse to the administration of either prophylactic serums or prophylactic drugs. However, the prophylactic use of either serums or drugs, or both, together may serve a valuable purpose in making both early and delayed surgery more secure, or in permitting primary closure of a wound which would otherwise be left open, and many surgeons now prefer to use both these adjuncts. But the use of serums or drugs can never be regarded as a substitute for adequate surgery.

As a bacteriologist, the writer is impressed with the general lack of careful, detailed bacteriology in most of the cases of gas gangrene that have been recorded. It seems important that more bacteriologists be trained in the technics of isolating and identifying the bacteria, particularly the anaerobic bacteria, which occur in the mixed infections of malignant edema and gas gangrene. It seems obvious, that too many of those who report these cases feel that they have done their duty when they record the presence of "the gas bacillus." It is rare, indeed, to see a detailed report of the various aerobic and anaerobic species which are generally present.

The treatment of infected wounds under catastrophic conditions, as in war, earthquakes and train accidents, must, necessarily, be based upon the assumption that prompt surgery is generally impossible. Under these conditions the injection of polyvalent, preferably pentavalent, gas gangrene antitoxin and tetanus antitoxin assumes a major prophylactic rôle, making possible and effective delayed surgery to save lives and limbs. It is necessary, of course, to guard against anaphylactic shock in asthmatics and others sensitive to horse serum.

Sulfonamide drugs and penicillin may serve a similar prophylactic rôle. Porter[105] was quite convinced that the value of application of sulfanilamide in war wounds and of sulfathiazole by mouth was fully proved. Key[106] believed that sulfonamide powders should be implanted in all clean operative wounds in both civilian and military hospitals, but Meleney's[107] observations on civilian wounds cast serious doubt upon this prevalent practice. Meleney found from his analysis of 1,500 civilians with contaminated accidental wounds, compound fractures and burns, that while the use of the sulfonamides minimized the general spread of infections and cut down the incidence of septicemia and death, there was no evidence that they lessened the incidence of local infection. One can only point out that the conditions under which most civilian wounds are treated are so different from those under which war wounds are treated that it is scarcely possible to reason from one to the other. The final judgment in reference to the prophylactic value of both antitoxic serums and drugs, applied either locally or systemically to war wounds, would seem to this writer best decided by similar statistical studies upon war wounds.

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THE IMMEDIATE AND LATE TREATMENT OF AN ARTERIOVENOUS FISTULA*

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ONE MAY PREDICT with confidence that many abnormal communications between the larger vessels will be established by wounds sustained in this war and that they will provide, unless early and successful treatment is available, some very interesting clinical phenomena and difficult problems both for the internist and the surgeon. In 1937, Basil Price[1] recorded his experiences with five cases from the last war whose arterial-venous wounds were sustained 20 years previously. In three instances operations had been considered impossible because of advanced cardiac disability, and two patients had died, one from a ruptured varicose vein distal to the fistula, and one from cardiac failure. The third patient will undoubtedly die of cardiac decompensation unless an operation for the elimination of the fistula can be performed successfully, a possibility which should still be considered despite the cardiac disability. Even advanced cardiac failure with generalized edema, ascites and hydrothorax has been cured completely by the elimination of an arteriovenous fistula.[2] In Price's two remaining cases incomplete operations have improved but have not cured the lesions. Increasing cardiac disability directly due to the fistulae may be expected as time elapses, and reoperation should also be considered for them, if at all possible.

The lesion provides one of the most fascinating examples of pathologic physiology:[3] the introduction of a fistula into the circulation superimposes upon the normal circulatory bed a second, or fistulous, circuit which is in reality *parasitic* upon the first. The normal circulation consists of the heart, arterial bed, capillary bed, and venous bed. The fistulous circuit consists of the heart, the artery between the heart and the fistula, the fistula, and the vein between the fistula and the heart. Common to both systems, are obviously, the heart, the artery to the fistula, and the vein from the fistula to the heart (Fig. 1). Each system requires a certain volume of blood to satisfy its needs, the need of the fistulous circuit depending upon the amount of blood which the fistula is capable of transmitting. Experimentally, and clinically, when the parasitic circulation attracts to it the greater volume of blood, the animal or patient dies.

The effect of the diversion of blood from the normal arterial bed into the parasitic circuit is many fold: There occurs (1) a lowering of blood pressure comparable with that accompanying massive external bleeding from a large vessel; (2) an accelerated pulse rate as a compensation for the lowered blood pressure, and as a response to the increased venous filling of the heart;

*The opinions contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

(3) a great increase in cardiac output indicating a greatly increased volume flow of blood through the fistulous circuit; (4) a temporary or fleeting reduction in the size of the heart, and of the artery proximal to the fistula, due to a redistribution of blood from the central arterial to the peripheral venous vascular bed.

The loss of blood from the normal circulation with its concomitant fall in blood pressure is at first rectified, as in any bleeding to the outside, by the accretion of fluid from the tissues, and by cells and fluid from such organs as the spleen and liver. This restoration of the circulating blood to a volume adequate to meet the needs of both the normal and parasitic circulations results in (1) a recovery of systolic blood pressure to normal, but a permanent lowering of diastolic pressure due to a permanently decreased peripheral resistance at the site of the fistula; (2) a return of the pulse rate to a more normal rate, although at times the acceleration may persist; (3) an increased total blood volume; and (4) an increase in the volume of blood flowing through that part of the circulatory system common to both the normal and parasitic circuits.

This increased volume, or bulk, of blood flowing through the fistulous circuit including the four chambers of the heart, distends and dilates it to a greater or less degree depending on the volume flow through the fistula. Experimentally, it has been proven that the entire part of the circulatory bed through which the short-circuited blood passes becomes dilated.[4]

The interdependence of the size of the normally functioning heart and the volume flow of blood through it has been demonstrated also under other conditions. In massive bleeding the removal of 500 cc. of blood from the circulation of a 12-kilogram dog will reduce the heart to one-half its normal size. In shock the size of the heart is greatly reduced due to redistribution of the normal blood volume. In histamine shock the peripheral vasodilatation causes the blood to leave the central circulatory bed and to accumulate in the peripheral vascular bed. In traumatic shock, the blood leaves the central bed, and accumulates in the traumatized area. Similarly, on opening a fistula the blood leaves the normal arterial bed, and fills the capacious, easily distensible venous bed, and the heart temporarily decreases in size. Experimentally, however, this initial decrease in the size of the heart is very fleeting, lasting perhaps a few hours to a few days, after which there occurs a gradual dilatation of the heart and of the vessels leading to and from the fistula, in some instances to an enormous size.

This gradual dilatation of the fistulous circuit which often continues over a period of years, is explained as follows: The peripheral resistance at the site of the fistula in circuit PN (Fig. 1) is obviously less than the peripheral resistance in the arteriolar and capillary bed of circuit N. Flowing blood, like flowing water, seeks the path of least resistance. The tendency, as long as a difference in peripheral resistance persists, is to force at each heart beat a little more blood through the fistula at the expense of the blood flowing through the normal capillary bed N. But the blood loss from

circuit N is promptly rectified by an increase in the total volume of circulating blood, resulting inevitably, though slowly, not only in a gradually increasing volume of blood flowing through that part of the circulatory bed common to both but also in its dilatation. The determining factor in this gradual dilatation is the extent of the difference in the peripheral resistances in the two circuits, which, in turn, is determined by the size of the rent in the artery producing the fistula; by its location in the arterial tree; and by

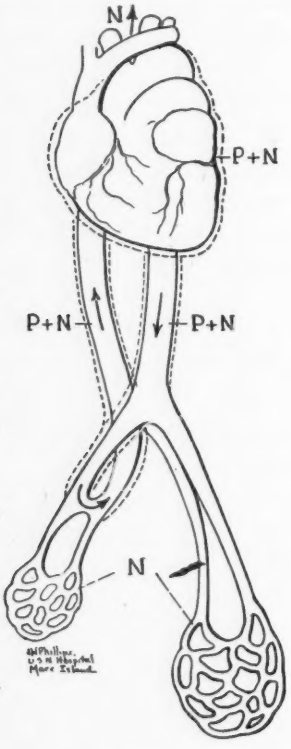

FIG. 1

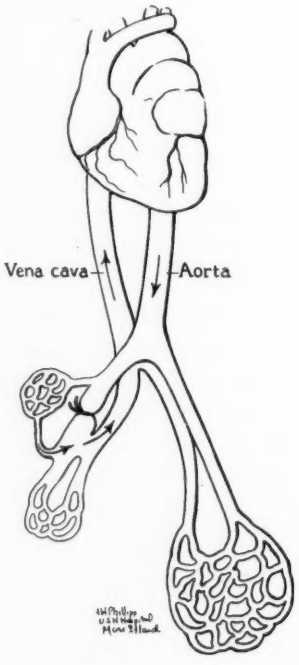

FIG. 2

FIG. 1.—The introduction of a fistula into the arterial bed introduces a *parasitic* circulation P which abstracts a certain volume of blood from the normal circulation N. The part of the circulatory bed transporting the two circulations P and N will dilate to accommodate the increased volume of blood coursing through it.

FIG. 2.—Ligation of the artery alone, such as might be undertaken proximal to a simple aneurysm, is absolutely contraindicated proximal to an arteriovenous fistula. It is obvious that the blood flowing through the collateral vessels would flow back to the heart through the site of the low resistance at the fistula, thus by-passing the peripheral vascular bed. Gangrene would under these conditions be inevitable, hence the importance of differentiating a simple aneurysm from an arteriovenous fistula.

such fortuitous conditions as the amount and firmness of the fibrous tissue deposited in the process of healing around the fistula, around the vein proximal or distal to the fistula, and around the artery proximal to the fistula. If this fibrous tissue is considerable, and the fistula is small, a point will be reached rather promptly when the peripheral resistances in the two circuits will become equalized, when there will be no further increase in the volume of blood flowing through the fistula, and when no further dilatation of the

fistulous circuit will occur. Such equalization may be indefinitely postponed when the fistula lies between vessels that offer little resistance to their dilatation as in the abdomen, pelvis or upper thorax, or when the fistula itself has a minimal amount of fibrous tissue deposited around it permitting its easy though slow dilatation. The slowly progressive dilatation of the heart that occurred in a few clinical cases observed 24, 25, and 26 years[5] after the inception of the fistulae is explicable only on the basis of this slow dilatation of the fistula itself, producing in effect a vicious circle. The extent of the fibrous deposits around a fistula and the involved vessels may determine the great variation in the effects upon the circulation of the fistulae located in the same general region in the vascular bed. If (by chance) an injury to the vessels is accompanied by a mechanical block in the proximal vein, thus preventing an easy return flow to the heart, the effect upon the circulation and upon the heart will develop much more slowly than in the absence of such a block. Mason[6] records cardiac decompensation in a subclavian fistula within nine weeks of its inception, whereas, in a patient observed by me, seven years elapsed before cardiac decompensation appeared in the presence of a subclavian-jugular fistula.

A most important factor in determining the effect of a fistula upon the circulation is its location in the arterial tree. The nearer the fistula is to the heart, and the larger the vessels involved, the greater will be the discrepancy between the high pressure in the artery, and the absence of pressure in the vein, the flow of blood through the fistula being commensurate with this difference in arterial and venous pressures. As a corollary to this it may be said that the larger the injured artery, the greater will be the capillary bed normally supplied by this artery, and, therefore, the greater will be the peripheral capillary resistance beyond the point of injury or communication with the vein. Obviously, therefore, the extent of the difference between the negligible resistance in the fistulous circuit PN (Fig. 1) and the peripheral resistance in the vascular bed N beyond the fistula will be determined by the location of the fistula in the arterial tree, which, in turn, will determine not only the amount of blood diverted through the fistula but the resulting effect of the fistula upon the circulation. A fistula 15 mm. long, established between the aorta and vena cava in a dog as a lateral anastomosis, is almost invariably fatal immediately, whereas, a fistula 15 mm. long, established as a lateral anastomosis between the femoral vessels, will produce cardiac dilatation and decompensation only after years have elapsed.

Other factors influence the ultimate effects upon the circulation. If the tangential wound in the artery produces a fistulous opening larger than the proximal artery, and is, therefore, capable of transmitting more blood than this artery can supply, the arteries in the collateral bed surrounding and proximal to the fistula will open up to pour their quota into the parasitic circuit, to satisfy, as it were, its thirst for blood. This stimulus to the development of the collateral circulation is entirely due to the ease of flow through the site of decreased resistance at the fistula, and is not dependent

upon the need of the tissues beyond the fistula, as Reid,[7] and Lewis[8] have suggested.

The very occasional and rare dilatation of the artery *distal* to a fistula[9] is easily duplicated in the experimental animal by establishing a fistula and then tying the artery proximal to the opening. The site of diminished peripheral resistance at the fistula attracts blood from all available collateral arteries connecting with the branches of the artery distal to the fistula. The resulting greatly increased volume of blood flowing back through the distal artery into the fistulous circuit produces a dilatation of this artery distal to the abnormal communication. These experimental observations demonstrate the intimate relationship between the decreased peripheral resistance to the flow of blood through the fistula and the development of collateral circulation, and the intimate relationship also between the volume flow of blood through an artery and its resulting dilatation. Clinically, obstruction of the artery proximal to a fistula may occur due to fibrous tissue deposited in the course of healing, and, as a result, dilatation of the artery distal to the fistula may be observed.

Closing a fistula, either by digital compression or by operation, results in a reversal of all these various effects upon the circulation: the elimination of the parasitic circulation directs the blood formerly short-circuited into the fistulous circuit into the general arterial bed, distending it with a volume of blood abnormally increased in the presence of the fistula. The distension of the arterial system includes not only the already dilated heart, which temporarily becomes even more dilated, but also the arch of the aorta whose depressor fibers of the vagus nerve are stimulated producing a reflex retardation in pulse. This is a protective and natural response to the abnormally high blood pressure incident to the abnormal distension of the arterial tree. Experimentally and clinically, it has been shown that the increase in blood pressure precedes the retardation in pulse, both phenomena being dependent upon the increase in total volume of blood. There is no other possible explanation for these phenomena of increased blood pressure and retarded pulse rate on closing a fistula except on this basis of an increase in total blood volume, and no other tenable explanation has been offered by those authors reluctant to accept the clinical and experimental demonstration of this increase in total blood volume.

The overdistension, high blood pressure and retarded pulse rate following permanent elimination of a fistula are promptly rectified by a reduction in total blood volume through loss of blood plasma, as shown by an excessive urinary output and by a concentration of blood in the first 24 hours after closing a fistula. As a result of this decrease in total blood volume and of the diminution in volume flow of blood through the fistulous circuit, there occurs a rapid decrease in the size of the heart, and except for a very slight hypertrophy that has occurred during the life of the fistula, the heart will be restored practically to normal size within six to ten days.

It is obvious from the foregoing observations that a fistula large enough

to produce circulatory changes must be eliminated from the circulation to avoid a slowly progressive cardiac disability from cardiac overdistension, a complication that may occur promptly after the introduction of the fistula, or as much as 25 to 30 years later.

The following sequence of clinical events usually accompanies a fistula: dyspnea and tachycardia on the slightest exertion; an increasingly vigorous beating or "pounding" of the heart; and a progressive dilatation of the heart, followed inevitably by complete invalidism and death. There may also follow in the wake of the fistula various local manifestations, such as edema of the extremity, often elephantiasic in type, marked varicosities complicated by eczema, ulceration and occasionally bleeding.

A patient in whom an injury to a large artery is suspected is placed in a hospital for continuous and careful observations. If, under bed rest, the bleeding ceases, and there is no increase in the swelling of the limb due to an enlarging hematoma, nor evidence of developing infection, further delay in operating is advisable. An increasing hematoma at the site of the injury, or an increasing swelling of the limb which threatens the blood supply of the extremity beyond it, as shown by a cold, edematous, and pulseless leg, or arm, requires immediate operation. The operation at this stage is undertaken preferably under a tourniquet, if feasible to apply it, the hematoma is evacuated, a localized débridement is performed, and the injured vessels exposed. In the absence of any evidence of infection, and if the arterial wound is small and unaccompanied by loss of substance, a suture of the wound may be attempted. The vein is ligated to avoid the danger of an embolus either of air or blood clot. If such suture is impossible, the ligation and division of artery and vein are in order. Repeated and massive transfusions are indicated to maintain and raise peripheral arterial pressure, since this is the best guarantee that a limb deprived of its main artery will survive. Interruption of the lumbar or thoracic sympathetics either by repeated procaine injections, or by direct operation, will encourage collateral circulation by promoting vasodilation.

Should evidence of infection be present, the hazard to life is greatly increased. Under a tourniquet, if feasible, the wound is laid widely open, blood clots are removed, a débridement is performed, the injured portions of the artery and vein are excised, and the four ends of the vessels are ligated. Every nook and cranny of the wound is liberally treated with sulfathiazole powder, smeared in as an emulsion or suspension in tissue fluids, and sulfonamides are given by mouth. Penicillin is administered in maximum dosage.

If the wound to the artery appears trivial, and the diagnosis is not immediately made (and this is often the case), operation should be deferred until all danger of infection is past. Indeed, the observation that small fistulae heal spontaneously has led Reid[7] to advocate postponement of surgical intervention for six months. Reid emphasized also that delay in operating upon a recently formed fistula is indicated, so that the collateral circulation may be developed sufficiently to permit division of the artery if necessary. This

delay may be as short as five to six weeks, or as long as four to six months, without the life of the patient being endangered. Other benefits of delay suggested by Reid are that the injured vessels become more thoroughly healed, thus, making their dissection easier and safer, and infection is less likely to occur. For these various reasons, unless cardiac decompensation is imminent, it would be preferable to delay operation upon an arteriovenous fistula for five to six months, at the end of which time an operation may no longer be necessary. Should cardiac disability manifest itself at any time before the elapse of six months, operation should be undertaken at once. Experiments have shown that although small fistulae tend to heal spontaneously, large fistulae do not. If, therefore, evidence develops that the heart is enlarging, that the thrill and bruit are increasing rather than diminishing in intensity, that variations in blood pressure and pulse can be produced by closing the fistula, and that these variations are becoming more, rather than less, pronounced one may be certain that the opening will not close spontaneously and that it must be eliminated by operation to avoid further ill effects upon the circulatory system.

An important preoperative precaution in a fistula exhibiting cardiac disability is to prescribe complete rest in bed for 10 to 14 days preceding the operation. Digital closure of the fistula, or of the artery proximal to the communication, for 30 to 40 minutes three to six times daily will be very helpful in controlling or improving the cardiac disability. It acts, I believe, by reducing the amount of blood flowing through the fistula by encouraging fibrous contraction about the fistula. Several patients were greatly improved before operation by this simple expedient.

Following the operative closure of a large fistula which has produced a marked dilatation of the heart, it is important to restrict activity for six to eight weeks to permit the previously dilated and thinned-out cardiac musculature to become readjusted to the increase in diastolic pressure brought about by closure of the fistula. An important precaution at the operating table is also related to this extreme dilatation of the heart accompanying a large fistula. Closure of the fistula not only raises diastolic pressure by an increase in peripheral resistance, but also leads to an overdistension of an already dilated heart through a redistribution of the circulating blood, more than half of which formerly leaked into the capacious venous bed. Such overdistension of an already thinned-out cardiac muscle might easily lead to recurrence or exacerbation of the cardiac decompensation. This would be revealed at the operating table by an increased pulse rate and a lowered blood pressure on closure of the fistula, instead of the reverse. Should this occur the operator should be prepared to perform an immediate venesection in order to reduce the volume of circulating blood which had become considerably augmented during the life of the fistula.

In operating upon an arterial lesion certain fundamental principles must be observed. If a venous communication is overlooked, and the usual hunterian ligation of the artery proximal to the lesion is performed, gangrene

beyond the fistula is almost inevitable. The collateral circulation will find its way not into the capillary bed distal to the fistula, but through the fistula back to the heart (Fig. 2). Hence, the importance of accurate observations to determine whether the lesion is a simple sacculated aneurysm or an arteriovenous communication, since life itself may depend on the correct differentiation between these two conditions. The distinctive features of a fistula as compared with a simple aneurysm are: (a) the thrill and bruit are continuous but intensified during systole; (b) the slowing of the pulse and rise in blood pressure on digital closure of the artery proximal to the lesion occur only in the presence of a fistula and never in the presence of a simple arterial aneurysm, since total blood volume is not increased in the presence of the simple aneurysm; (c) the high oxygen content of arterial blood withdrawn from the veins distal to a fistula as compared to the venous blood obtained from another extremity is a distinguishing feature suggested by Brown.[10] If these evidences of a fistula are present, simple ligation of the artery proximal to the lesion is absolutely contraindicated.

The ligation of the artery *and vein* proximal to the fistula may occasionally be employed as a preliminary procedure in the hope that closure will be effected by thrombosis or by fibrous contraction at the site of the fistula. Usually the fistula is not cured, as the collateral channels will readily supply blood to the site of decreased resistance provided by the fistula. The ligation of the artery and restoration of the vein is definitely contraindicated, since the little blood that passes through the collateral channels will find its way promptly into the dilated venous bed, thus avoiding the distal arterial bed.

In certain appropriate cases, the Matas-Bickham[11] procedure of transvenous or transsaccular aneurysmorrhaphy may be attempted. Under a tourniquet, the varicose sac or the dilated vein is boldly opened, and the rent in the artery is closed by suture. The vein should be ligated above and below the fistula. The wall of the vein may be employed to reinforce the sutured rent in the artery.

Ligation of the artery and vein proximal and distal to the fistula with excision of the fistula is the operation of choice for complete cure, and the one usually most easily executed (Fig. 3). A pulsating artery that is full of blood is so much more easily identified than a collapsed vessel that isolation and mobilization of the vessels is best accomplished *without* a tourniquet, the artery proximal and distal to the fistula being isolated first for control in case of bleeding. If feasible, one should be prepared to apply a tourniquet at any time in the course of the operation.

Should complete mobilization and excision of the fistula be impossible because of involvement of important structures such as nerves embedded in dense fibrous tissue, ligation of the artery and vein proximal *and distal* to the communication without excision of the fistula is in order (Fig. 4). Under such conditions, however, the artery proximal to the fistula should be ligated *and divided,* to avoid reactivation of the fistula by the reopening of the artery through necrosis of the tissues included in the ligature.

Large pulsating aneurysmal swellings and arteriovenous fistulae of the upper thigh, above which the use of tourniquets is impossible, require careful planning and special measures lest incising the lesion engulf the surgeon and the operative site in a sea of blood, completely obscuring and defeating the object of the operation. An important maxim here, as elsewhere, is

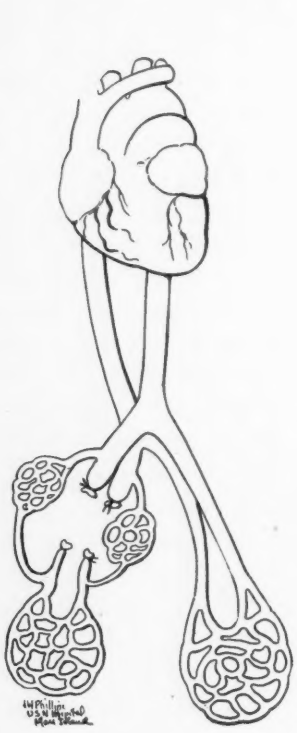

FIG. 3

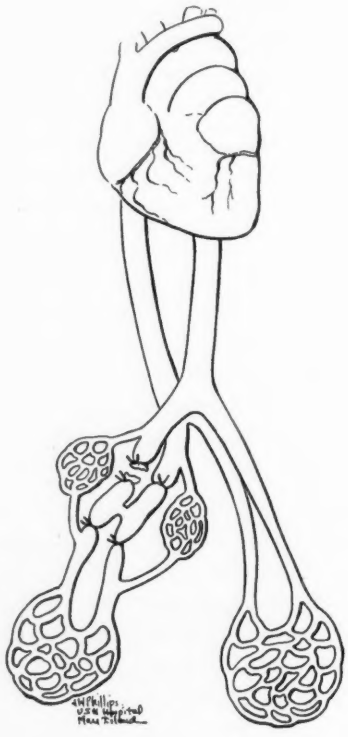

FIG. 4

FIG. 3.—The operation of choice for an arteriovenous fistula is ligation of the artery and vein proximal and distal to the fistula, and excision of the fistula. A fistula which has been present for four months or longer has usually so stimulated collateral circulation that ligation of the main artery can be performed with impunity.

FIG. 4.—If excision of the fistula is contraindicated because of difficulty in mobilization due to excessive scarring or to involvement of other important structures, such as nerve trunks, quadruple ligation, without excision, may be preferable. Under such conditions, however, the proximal artery must be sufficiently mobilized to permit ligation at two points and division of the vessel between the ligatures. Ligation in continuity invariably leads to reëstablishment of the lumen and reactivation of the fistula.

not to incise a pulsating swelling without first having attained complete control of the normal artery above, and preferably also below the lesion. Complete control of the bleeding when operating upon arterial lesions of the upper thigh demands that both common and external iliac arteries be temporarily occluded. The inclusion in the temporary ligature of a segment of rubber tubing about the caliber of the artery prevents fracturing the walls of the artery, which would invite later trouble at the site of temporary ligation (Gordon Watson[12]). It also enables one to remove the temporary ligature without difficulty and without injury to the artery by cutting the tape

ligature on the rubber tubing. Such temporary occlusion of the common and external iliac arteries is best accomplished through a separate incision through the tendinous structures of the abdominal wall just lateral to the lower third of the rectus muscle, displacing the peritoneum upward, and exposing the common iliac artery at its origin. The lesion itself is approached through a longitudinal incision in the thigh directly over the common and superficial femoral arteries. Division of Poupart's ligament is avoided if possible. This incision for the exposure of the common iliac artery will also permit removal of the lumbar sympathetic ganglia for the production of peripheral vasodilatation, if this seems desirable.

In operations upon the large vessels of the neck, the hazards of bleeding are also greatly accentuated. To operate successfully upon arterial lesions in this area, one must be able to expose the normal artery proximal to the arterial wound for temporary occlusion. One must be able, also, to occlude temporarily the proximal vein, not only to control bleeding but also to avoid air embolism through accidental rents in the larger veins which in this region so frequently exhibit negative pressure during inspiration. To insure an exposure adequate for operations upon the subclavian and axillary vessels, it has been found desirable and practicable to resect a considerable portion of the clavicle subperiosteally. No permanent damage has resulted from such resection, since retention of the periosteum has permitted reformation of the clavicle and restabilization of the shoulder within four or five weeks. The wide exposure of important structures permitted by resection of the clavicle provides greater confidence in being at all times master of the situation should any untoward event occur, such as unexpected arterial bleeding, tears in large veins, or injuries to the larger lymphatics. On the right side, the innominate artery and vein must occasionally be temporarily occluded by tape ligatures. Resection of the medial two-thirds of the clavicle and partial resection of the manubrium will reveal these underlying vessels.

Ligation of the common or internal carotid may be accompanied by hemiparesis of the opposite side, due to nutritional disturbances in the cerebrum. Due to a collateral flow through the external carotid artery, ligation of the common carotid is less dangerous than ligation of the internal carotid artery. Whenever ligation of either the common or internal carotid arteries must be undertaken, Makins[13] emphatically advises also occlusion of the internal jugular vein.

The success of operations upon the large vessels depends in great measure upon the avoidance of sepsis. The strictest precautions against infection must be followed throughout, and the liberal use of the sulfonamides locally and systemically is indicated in any contaminated or potentially infected wounds. Except in the presence of actual pus, drainage in vascular surgery must be scrupulously avoided. In surgery of the large vessels, the packing or drainage of wounds is inviting almost certain disaster. Should the wound at any time following operation fill up with fluid, it is a simple matter to evacuate it under strictly sterile precautions. If there is any question of

infection, penicillin should be promptly administered in maximum dosage. If drainage of a contaminated wound seems desirable, line the wound with gauze heavily impregnated with petrolatum, but avoid touching the site of ligation or the line of suture in the artery by any foreign body such as the petrolatum drain. A drain lying against the site of ligature interferes with the protective deposition of fibrin and fibrous tissue around the ligature, and invites secondary hemorrhage through necrosis of the arterial wall at the site of ligation.

SUMMARY

1. The physiologic effects of an arteriovenous fistula are easily understood when the short-circuit to the heart is considered as introducing a new circulation *parasitic* upon the normal circulation.

2. The effect of such a parasitic circulation is dependent entirely upon the amount of blood transmitted to it through the abnormal communication, which, in turn, is dependent upon numerous factors: the actual size of the opening, the size of the artery, its location in the vascular tree which determines the extent of the difference between end-pressure in the artery and the absence of pressure in the vein. Further factors determining the amount of blood transmitted through the opening are such fortuitous ones as how much unyielding fibrous tissue has been deposited around the fistula, around the artery, and around the vein both proximal and distal to the fistula.

3. Immediate operation for a fistula may be necessary if the wound in the artery is accompanied (a) by uncontrollable bleeding; (b) by progressive enlargement of the pulsating hematoma of the soft tissues; or (c) by progressive interference with the development of collateral vessels by swelling of the limb through infiltration of tissues with blood under arterial pressure.

4. Operation should be postponed for five to six months if the conditions noted above permit it, because:

(a) A small fistula may heal spontaneously.

(b) Collateral circulation should have an opportunity for development in case the main artery to the limb must be ligated.

(c) Tissues infiltrated with blood will return to normal, permitting better identification of structures and easier dissection of tissues.

(d) The danger of infection is reduced.

5. Dissection of vessels is more easily accomplished if they are full and pulsating; hence, operation *without* a tourniquet is preferable whenever feasible. Control artery and vein proximal and distal to fistula in good tissues first.

6. In fistulae of the neck, exposure of normal vessels proximal to the fistula is absolutely necessary, and greatly facilitated by subperiosteal removal of the clavicle, and, on the right side, by removal of portion of the sternum to expose the innominate artery and vein if indicated.

7. In fistulae of the upper thigh, particularly when associated with false

aneurysmal sacs, both common iliac and external iliac arteries must be controlled with temporary ligatures (including segment of rubber tubing to avoid injuring the arterial wall) before incising sac or exposing the fistula through a pulsating hematoma.

8. The iliac vessels are best exposed through a separate incision paralleling and along the lateral border of the lower one-third of the rectus muscle, the femoral vessels being exposed through a separate longitudinal incision paralleling these vessels. The incision should preferably not cross the inguinal ligament.

9. The operation of choice for an arteriovenous fistula is ligation of artery and vein proximal and distal to the fistula and excision of the fistula following careful isolation.

10. If excision of fistula is impossible because of fibrous deposits and adherence of important structures, such as nerves, not only ligate artery and vein proximal and distal to the fistula, but also divide proximal artery between two ligatures to avoid subsequent reestablishment of fistula.

11. Under no circumstances ligate the artery alone proximal to an arterial lesion presenting evidence of a continuous bruit indicating a venous communication. Gangrene is inevitable following such ligation.

12. Transvenous or transsaccular aneurysmorrhaphy, with ligation of vein proximal and distal to the fistula (Matas-Bickham procedure), may be employed in selected cases if the tourniquet can be applied above the lesion.

13. Drainage in vascular surgery is rarely necessary, but if used great care should be exercised to avoid touching the site of ligation or of suture by the drain. Such a contingency increases the possibility of secondary hemorrhage.

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ARTERIAL INJURIES IN A THEATER OF OPERATIONS

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A GENERAL HOSPITAL in a Theater of Operations receives a variety of arterial injuries. Battle-incurred arterial injuries are usually dealt with in Forward Hospitals, but not infrequently these injuries are not apparent until the patient has been evacuated to the Base, usually a matter of a few days. Arterial injuries seen at the Base may be divided as follows:

I—Traumatic vasospasm, without laceration of the artery.

II—Direct arterial injury:

A. Early results of injury.

B. Late results of injury.

III—Pulsating hematoma (traumatic aneurysm).

IV—Arteriovenous aneurysm.

I—Vasospasm is a protective response to trauma, but if the arterial tree of an extremity becomes, and remains, constricted, then thrombosis, trophic changes, fibrosis and lesions resembling Volkman's ischemic contracture may follow. It has been pointed out by DeBakey,[1] and Elkins,[2] that remote trauma may result in complete spasm of an artery. We have observed 12 patients with persistent posttraumatic vasospasm in which the arteries were not directly injured. The involved extremities were cold, cyanotic and often edematous. Vasospasm could be temporarily abolished by paravertebral injections of novocaine (1 per cent). In the more severe cases, preganglionic sympathectomy was performed, with good result. The following case history is typical of this group:

Case Report.—A 28-year-old, white, male, received shell fragment wounds of the right upper leg and right lower thigh in February, 1944, near Cassino, Italy. Fifteen hours later, at an Evacuation Hospital, the wounds were débrided and the shell fragment removed from the leg through an elective incision along the head of the fibula. Exploration of the posterior tibial artery showed it to be in marked spasm, but the popliteal artery could be felt to pulsate. Lumbar paravertebral injection was done immediately, as the foot was cold, with absent peripheral pulses. Three more injections were done within the next few days. The patient was admitted to this hospital 13 days after injury. The right foot was colder than the left, slightly swollen and the peripheral pulses were questionably palpable. The wounds were closed two days after admission, and it was necessary to repeat the paravertebral "block" as the foot became cyanotic and clammy. The wounds healed normally, but the foot continued clammy and cyanotic, becoming warm and pink only after "blocks." Femoral pulsations were normal, but neither the popliteal nor peripheral pulses could be felt. In all, eight paravertebral novocaine injections were done. A month after injury preganglionic sympathectomy was performed. The foot became flushed and warm in eight hours, and the peripheral pulses remained palpable.

Other patients in this group show that posttraumatic vasospasm may persist over long periods of time, and may be either segmental or may involve the entire arterial tree of the extremity. If vasospasm is not eliminated by a series of paravertebral "blocks," we feel that sympathectomy should be considered.

II—Direct Arterial Injuries.—A—*Early Results:* Direct trauma to major arteries usually leads to thrombosis at the site of injury, formation of a traumatic aneurysm, or formation of an arteriovenous fistula. The latter two were combined in one of our cases.

Ligation of the lacerated artery (and usually its concomitant vein) at the time of the initial débridement is the procedure usually carried out in most instances. The records show that few attempts at repair or end-to-end anastomosis have been attempted in the Forward Hospitals. Captain Pryor,[3] in studying 361 major extremity amputations performed or admitted in this hospital, found that approximately 20 per cent were done because of inadequate blood supply alone. A study of 55 patients whose arterial injuries resulted in amputation showed almost half of them (25) were due to popliteal artery injuries alone and 18 followed femoral artery injuries. In all, we have observed 29 patients whose popliteal artery was ligated. Twenty-five of them had gangrene, which required amputation (86 per cent). It may be significant that of the four whose extremities remained viable, three had lumbar ganglionectomies, and the other one had repeated paravertebral injections. It is recognized that other factors may be of greater importance in determining viability of the leg.

Our experience with the nonsuture anastomosis of blood vessels, suggested by Blakemore, Lord and Stefko,[4] has been limited to three patients, upon whom the procedure was done before admission to this hospital. Two of these soldiers had a viable foot, but the third developed dry gangrene of all toes. Since the anastomosis was undertaken because of laceration of the popliteal artery, we feel that the result obtained was better than that usually effected without the venous anastomosis. Peripheral pulses were not palpable in any of the three extremities, and arteriograms have shown that the medium did not go through the anastomosis.

In an effort to decrease the number of amputations secondary to arterial injury, sympathectomy has been performed upon eight patients (Table I). Two of them with popliteal ligation eventually required amputation through the leg. These two patients had been injured for five and four days, respectively, and had not received paravertebral injections. Three other patients had sympathectomy within 48 hours after popliteal artery injury, and the extremity remained viable. This series is too small for any definite conclusions, but the results suggest that early sympathectomy in major arterial injuries may help preserve a part, or all, of an extremity.

B—*Late Results:* The late results of major arterial ligation, also, deserve consideration. In a series of seven patients whose femoral or popliteal artery had been ligated, there was evidence of chronic arterial deficiency. This was characterized by intermittent claudication on walking, "muscle cramps" at rest, weak to absent peripheral pulses and trophic skin changes, as well as comparative coolness of the extremity. Preliminary paravertebral injections showed a favorable response, and sympathectomy gave excellent results (Table II). Bigger[5] recently reported on 29 patients with aneurysm,

TABLE I

LUMBAR SYMPATHECTOMY IN "EARLY" ARTERIAL INJURIES

Case No.	Age of Patient	Preoperative Findings	Interval v.s. Injury and Sym.	Operative Result	Remarks
1	31	Ligation com. femoral. D/3 leg cold. Gangrene of 2nd toe	6 days	Probably unchanged	Supracondylar amputation required 48 hours later, as the entire gastrocnemius appeared necrotic. Extremity had been elevated on 4 pillows prior to admission.
2	34	Comp'd fracture tibia and fibula. Early gangrene of toes, with cold foot	13 days	Improved	Foot became warmer following operation, but amputation at the ankle was done 10 days later for the preexisting gangrene.
3	47	Ligation popliteal artery. Cold, discolored foot	4 days	Little change	Gas gangrene developed in the leg wound on the 6th day after injury, and amputation was done. Extensive thrombosis of vessels throughout the leg was found on dissection.
4	28	Ligation popliteal and comp'd frac. fibula. Gangrene great toe	5 days	Leg became warmer	Amputation lower 3rd of the leg was done 3 days later because of fever, necrosis of gastroc. and soleus muscles. It was felt that sympathectomy probably permitted amputation at a lower level.
5	19	Ligation com. femoral. 4 lumbar "blocks." Cold, pulseless foot	2 days	Excellent	Foot became suddenly warm about 40 hours after operation, and remained as warm as the normal one.
6	20	Ligation popliteal. 1 lumbar block. Comp'd fracture head of fibula	1 day	Fair	Skin gangrene of tips of two toes developed, but foot remained warm.
7	19	Laceration popliteal artery and vein. Foot pulseless	8 hours	Good	This patient also had a nonsuture anastomosis, using segment of saphenous vein and vitallium cuffs (Blakemore).
8	27	Ligation popliteal. Foot cold	1 day	Good	Foot remained warm and viable.

TABLE II

LUMBAR SYMPATHECTOMY IN ARTERIAL DEFICIENCY

Case No.	Age of Patient	Preoperative Findings	Interval v.s. Injury & Sym.	Operative Results	Remarks
1	27	Ligation Common femoral. Angina crisis. Cold foot	34 days	Improved	There was marked increase in the warmth of the foot and leg and decrease in the cramping.
2	22	Ligation superficial femoral. Cool foot	30 days	Excellent	The foot remained much warmer than the uninjured extremity. Patient's walking range was greatly increased.
3	21	Ligation common femoral. Intermittent claudication	71 days	Excellent	Can walk about ten times as far, before cramping occurs.
4	21	Ligation superficial femoral. Claudication after 200 yards	86 days	Excellent	Foot remained much warmer, and claudication disappeared.
5	29	Ligation of popliteal comp'd fract. femur. Cold, swollen foot	11 days	Excellent	Preoperative "blocks" caused flushing of the foot. Postoperative, the foot could be elevated in skeletal traction without blanching as before operation.
6	22	Ligation femoral vessels. Claudication on walking	68 days	Excellent	Walking range greatly increased. Patient returned to duty.
7	20	Ligation popliteal vessels. Daily lumbar blocks for 6 days	19 days	Good	Patient had developed a small area of necrosis on heel. Foot became much warmer, and wounds healed promptly.

or A-V fistulae, followed over a period of years. He found that a high percentage of these patients had chronic arterial deficiency when the main artery had been ligated. He suggests that sympathectomy may help relieve this deficiency.

III—*Traumatic Aneurysms.*—Traumatic aneurysms (pulsating hematomata) may not be apparent until some days after injury. When present, conservative measures have been carried out unless strong indications for early intervention exist. Such indications were present in about 40 per cent of our patients with aneurysms (Table III). The remainder were returned to the Zone of the Interior. In three instances, the expansile pulsation and systolic bruit previously present, spontaneously disappeared while the patients were awaiting evacuation.

TABLE III

TRAUMATIC ANEURYSMS

Artery Involved	Interval v.s. Injury & Oper.	Indications for Operation	Operation	Comment
Rt. axillary (3rd portion)	24 days	Increasing size of mass. Pain — Pressure on brachial plexus	Excision of artery involved. Ligation of ant. circumflex humeral. Ligation of axillary vein	Paravertebral (T 1, 2, 3) blocks with novacaine. Extremity warm. Normal union of clavicle.
Rt. axillary (2nd portion)	13 days	Severe secondary hemorrhage from aneurysm	Excision of artery involved with the "false" sac	Stellate ganglion block. Extremity warm and viable. Edema subsided in five days.
Rt. external carotid	20 days	Increasing pulsatile mass. F.B. of neck	Excision of artery involved with the "false" sac	No cerebral changes.
Lt. radial (midportion)	28 days	Superficial expansile tumor	Segment of artery and aneurysm excised *en masse*	Duty.
Rt. axillary (3rd portion)	5 hours	Tremendous hematoma. Absent radial pulse	Division and ligation axillary artery. Ligation of post. humeral and subscapular	Two paravertebral injections. Hand warm, and sensation and motion improved.
Rt. subclavian (3rd portion)	15 days	Undébrided wound of clavicle. Absent radial pulse. retained M.F.B.	Excision of lacerated segment. Subperiosteal resection medial ½ of clavicle	Paravertebral "block." Nail bed circulation good. Plaster yoke for three weeks.
Lt. superficial femoral	38 days	Increase in mass and severe pain in extremity 7 hours previously	Common femoral isolated through separate incision. Excision of lacerated segment and aneurysmal sac.	Preliminary lumbar sympathectomy. (8 days prior).
Lt. superior gluteal	8 weeks	Sudden increase in mass, with pain. Bruit had disappeared	Excision of aneurysm	No complications.
Lt. ext. carotid	10 days	Profuse hemorrhage into mouth	Ligation of external carotid, and plication of aneurysmal sac	No cerebral changes.
Rt. popliteal	2 hours	Hematoma. Cold, pulseless foot	Ligation and division of artery and vein	Multiple paravertebral blocks. F.C.C. femur treated with leg elevated only a few inches. Extremity viable.
Lt. common carotid	0.5 hour	Hematoma. Hemorrhage from stab wound	Ligation with transfixion sutures	No cerebral changes.
Rt. common carotid	8 days	Massive sec. hemorrhage. Aneurysmal sac	Excision of bifurcation of carotid, with ligation of common, int. and ext. carotid arteries	No cerebral changes.

Case 1.—This 25-year-old male had multiple, severe battle injuries, resulting in exteriorization of the hepatic colon, a compound spiral tibial fracture, hematoma of the right anterior aspect of the shoulder, and other extremity injuries. The hematoma was partially evacuated and a drain inserted through an axillary stab incision. Three days after admission to this hospital (10 days after injury) there was sudden increase in size of the right subpectoral mass and severe "burning" pain which involved the right upper extremity. Function of the hand and forearm became greatly impaired with hypesthesia over the radial and median nerve distribution. Blood pressure, right, was 104/76, left, 130/78. The mass was expansile and a systolic bruit was present. The pain became severe and required morphine at frequent intervals.

At operation (24 days after injury), the subclavian artery was temporarily occluded through a separate incision after dividing the clavicle. The aneurysm was then opened and the lacerated axillary artery divided between ligatures. The anterior circumflex humeral was also ligated at its origin at the site of injury. The hand remained warm, and motor and sensory function improved rapidly. Two paravertebral blocks were done postoperatively.

Case 2.—The indication for operation on this patient was severe secondary hemorrhage 13 days after injury. The false sac and segment of axillary artery involved were excised, and stellate ganglion block performed, but there was never any doubt as to the viability of the hand.

Case 3.—The expansile, pulsating mass in the neck steadily increased in size. The damaged portion of the right external carotid artery and false sac were excised without any complications.

Case 4.—This aneurysm involved the left radial artery, and following excision the patient was discharged to full duty.

Case 5.—This 23-year-old, white male was shot just above the right breast. On admission to this hospital, five hours later, there was an enormous hematoma of the right axillary and right pectoral regions, absent right radial pulse, and the hand was cold. There was no bruit. There was a 15-mm. laceration of the circumflex humeral arteries, so that ligation of the three vessels was necessary. The axillary artery was divided between ligatures. The distal stump pulsated feebly. The concomitant vein was also ligated. Paravertebral injections were performed postoperatively, and the hand remained warm and steadily improved in strength.

Case 6.—This 19-year-old, white, male, sustained a penetrating shell fragment wound of the right clavicular region, and was in profound shock when admitted to a Forward Hospital. There was a compound comminuted fracture of the clavicle. External hemorrhage ceased, and no operative procedure was carried out. There was paralysis of portions of the brachial plexus. On admission to this hospital, 12 days after injury, the wound of entry was infected, with spicules of clavicle exposed, absent radial pulse, but a viable hand. Penicillin administration was begun 48 hours before operation. The wound of entry was excised, and the medial half of the clavicle resected subperiosteally. The hematoma surrounding the subclavian artery was undergoing organization. The artery was found to be almost divided just lateral to the anterior scalene muscle. The artery was divided between transfixion ligatures. The distal stump pulsated slightly. The postoperative course was uneventful. A figure-of-8 plaster yoke was removed after three weeks. The radial pulse could not be felt but the hand was quite warm.

Case 7.—This patient sustained a penetrating shell fragment wound of the left midthigh. His record stated that the wound was débrided and the foreign body was not removed. A week later the patient had two secondary hemorrhages, and a large, pulsating hematoma was noted. On admission to this hospital an expansile mass the size of a grapefruit was present on the anteromedial aspect of the left upper thigh. There was a systolic bruit present. The foot was warm, though the peripheral pulses were barely palpable. The mass increased in size, and the patient complained of pain

in the thigh at all times. The second, third and fourth lumbar sympathetic ganglia and trunk were removed as a preliminary measure, since it was felt that the aneurysm might involve both the superficial and profunda femorals and the common femoral.

Eight days after sympathectomy the mass suddenly increased in size, and the pain became intense. The common femoral artery was temporarily occluded through a small, separate incision before exposing the large aneurysm. The aneurysm contained about 1,000 cc. of clotted and unclotted blood. The defect in the superficial femoral artery was too large for repair, so the entire segment just distal to the origin of the profunda down to a branch below the laceration was excised. There was bleeding from the distal stump. The vein was also sectioned between transfixion ligatures. The shell fragment was found in the clotted blood. The postoperative course was uneventful. The foot remained quite warm.

TABLE IV

ARTERIOVENOUS FISTULAE

Site of Fistula	Interval v.s. Injury & Oper.	Preoperative Findings	Operation	Comment
Post. tibial (M/3)	8 weeks	Machine-like murmur. Continuous thrill. Branham's sign	Quadruple ligation, and excision of fistula	Duty.
Post. tibial (M/3)	5 weeks	Machine-like murmur. Continuous thrill. Branham's sign	Quadruple ligation, and excision of fistula	Duty.
Lt. common carotid-jugular	5 weeks	Loud murmur; strong thrill; and large M.F.B. at site of fistula	Quadruple ligation, and excision of fistula	M.F.B. 1.5 x 1.5 x 1.0 cm found to lie in the aneurysmal varix sac. No cerebral changes.
Rt. axillary vessels (P/3)	9 weeks	Right brachial 80/70. Left brachial 130/70. Branham's sign — 18 min.	Quadruple ligation, and excision. Brachial plexus neurolysis	Proximal 2/3 clavicle resected subperiosteally. Hand warm. Pulsation distal axillary stump noted.
Lt. profunda femoris vessels	6 weeks	Machine-like murmur, and large aneurysmal sac	Ligation and excision of vessels. Evacuation of large hematoma	Patient had severe pain from false aneurysm. Good pulsation postoperative.
Lt. brachial vessels (M/3)	8 weeks	Murmur and thrill. Branham's sign. Radial pulse good	Ligation of vein and endo-aneurysmorrhaphy	Arteriogram showed artery to be patent postoperatively. Hand warm. Good radial pulse.
Rt. subclavian vein (1st portion)	8 weeks	Typical machine-like murmur, and thrill. Radial pulses equal	Excision varix proximal subclavian vein and adjacent scar tissue	No definite arterial communication with the dilated varix was found. Normal findings after operation.

Case 8.—This patient had multiple severe shell fragment wounds. A systolic bruit was noted over the left buttocks soon after admission to this hospital, but gradually disappeared. Two months after injury there was sudden pain and increase in the size of the mass. The aneurysm was excised.

Case 9.—This is the only instance in which the hunterian operation was carried out. The patient had a compound fracture of the mandible and secondary hemorrhage into the mouth. The intermaxillary bands were removed and pressure made over the carotid until the external carotid could be occluded. This checked the bleeding but the wound of entry was enlarged and plication of the aneurysmal sac carried out. There were no further complications.

Case 10.—This patient sustained a gunshot wound of the right lower thigh, with laceration of the popliteal artery and a compound fracture of the femur. On admission, two hours later, there was a large hematoma, and the foot was cold and pulseless. The popliteal artery was divided between ligatures after evacuation of the hematoma.

The popliteal vein was then ligated. Repeated paravertebral injections were carried out postoperatively. The foot became cadaveric in appearance on even slight elevation, but remained viable, and the fracture healed normally.

Case 11.—This 39-year-old, colored, male, was stabbed in the neck with a meat knife by another neuropsychiatric patient. There was profuse hemorrhage which was partially controlled by digital pressure. Under endotracheal anesthesia, the wound was enlarged, and the left common carotid found partially divided after the large hematoma had been evacuated. The artery was ligated. There were no cerebral complications during the next six weeks, and the patient was returned to duty.

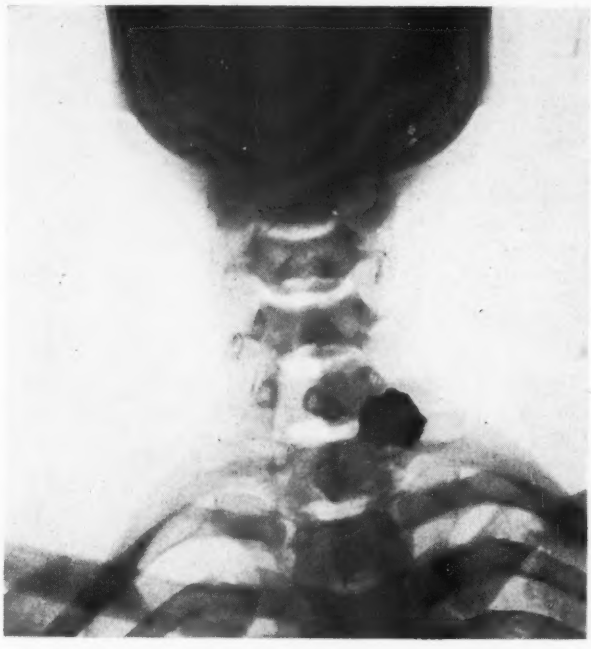

FIG. 1.—Anteroposterior roentgenogram.

Case 12.—This 30-year-old, white, male, had massive secondary hemorrhage into his mouth eight days after injury. There was an indurated, nonpulsatile mass in the right side of the neck, on admission, and Horner's syndrome was present. A small aneurysmal sac at the bifurcation of the common carotid was excised, necessitating ligation of the common carotid and the ends of the internal and external carotids. The excised segment showed marked atheromatous changes. There were no cerebral complications.

IV—Arteriovenous Aneurysms.—During the past year we have observed 13 patients with traumatic arteriovenous fistula. Seven of these were returned to the Zone of the Interior for definitive treatment later. Definite indications for operation were present in the remaining six (Table IV). There were no recurrences, gangrene, secondary amputations or deaths in any of the patients operated upon, for either aneurysm or arteriovenous fistulae.

It is important that the two conditions be differentiated, as both the local and systemic effects are different. An arteriovenous fistula is characterized

by a continuous machine-like murmur, which has been likened to the repeated whispered sound of the letter R. The palpable thrill or purr is also continuous. Branham's bradycardiac phenomenon is usually demonstrable when the proximal artery can be compressed.

Cases 1 and 2: Both patients had small arteriovenous fistulae involving the posterior tibial vessels. These were excised and the patients returned to duty.

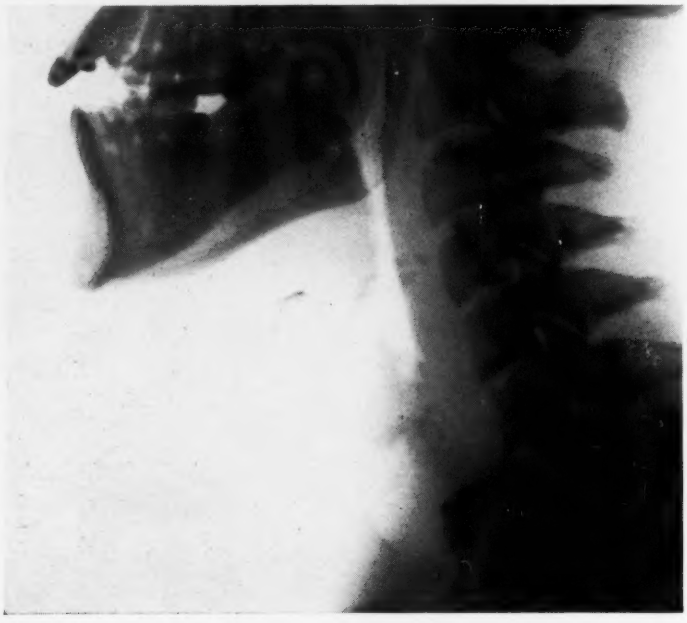

FIG. 2.—Lateral roentgenogram.

Case 3: This 20-year-old, white, male, sustained a penetrating shell fragment wound of the left infraclavicular region. A diagnosis of arteriovenous aneurysm was made 24 hours later (Figs. 1 and 2). On admission to this hospital, four days after injury, the wound of entry was healing. There was a very loud, continuous, machine-like murmur and thrill over the base of the left side of the neck. Pressure over the proximal vessels just above the manubrium caused the murmur and thrill to disappear. Brachial blood pressure determinations were equal. The murmur increased in intensity, but there was no evidence of cardiac enlargement. The patient was confined to bed because of extremity injuries as well.

At operation, a shell fragment, 1.5 x 1.5 x 1.0 cm., was found to lie within the aneurysmal varix sac, between the left common carotid artery and internal jugular vein. Quadruple ligation and excision of the sac and foreign body was carried out, as restoration of the artery was not possible. There were no postoperative complications (Fig. 3).

Case 4: This 26-year-old, white, male, sustained a perforating bullet

wound. The wound of entry was just below the medial third of the right clavicle, while the wound of exit was in the posterior axillary line. The right second rib and scapula were fractured by the bullet. The diagnosis was made, within 14 hours, at a Forward Hospital. On admission to this hospital, three days after injury, the thrill and bruit characteristic of an A-V fistula were present in the right infraclavicular space. Blood pressure, left

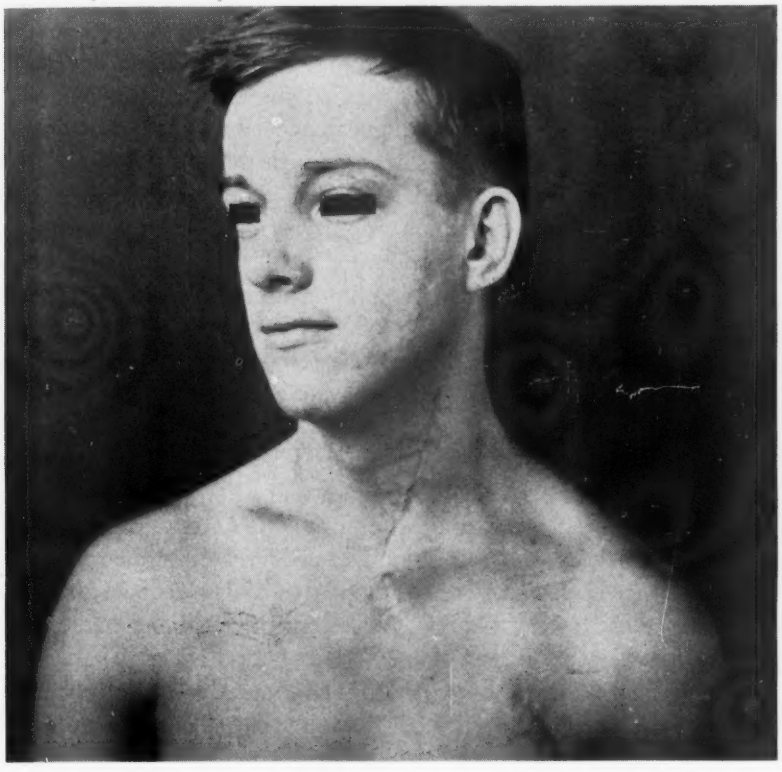

FIG. 3.—Ten days after quadruple ligation and excision of a common carotid-internal jugular fistula.

brachial 130/70, and right 80/70. The pulse rate slowed 18 beats per minute on compression of the proximal subclavian artery. There was some median nerve involvement. There was a progressive increase in the size of the heart shadow, though it remained within the upper limits of normal.

At operation, nine weeks after injury, the medial two-thirds of the clavicle were resected subperiosteally, to permit exposure of the vessels proximal to the fistula. The fistula was dissected out of a mass of scar tissue after the vessels had been secured both proximally and distally. Several trunks of the brachial plexus were freed from scar tissue and a small, superficial component of the median nerve was found to be divided and was sutured. The distal axillary stump pulsated. Convalescence was uneventful.

Case 5: This 28-year-old, white, male, sustained a shell fragment wound

of the anterolateral aspect of the left upper thigh. The missile traversed the left thigh, passed through the base of the scrotum, then the right thigh, fracturing the right femur. The wound of exit was on the posterolateral aspect of the right thigh. A large hematoma of the left thigh was evacuated at time of débridement.

On admission, a week after injury, the patient had a large mass in the anteromedial portion of the left thigh, which was slightly expansile, and quite tense. There was also a continuous, machine-like murmur of the upper left thigh and palpable thrill, which disappeared on compression of the common femoral artery. Skeletal traction was applied for the reduction of the right femoral fracture. A diagnosis of traumatic aneurysm and an arteriovenous fistula was made. The patient had severe pain in the left thigh, and burning and hypesthesia of the left foot and leg. The pain could not be controlled without morphine, therefore, operation was performed six weeks after injury. The foot was quite warm. At operation, the fistula was found to involve the profunda femoral vessels as well as communicating with an aneurysmal sac which contained over 1,000 cc. of old blood. The fistula was excised and the aneurysmal sac evacuated. The superficial femoral artery remained intact, so the circulation was adequate. Traction was continued until firm union of the femur was obtained.

Case 6: This 31-year-old, white, male, received multiple penetrating and perforating shell fragment wounds of the trunk and extremities. He was admitted to this hospital one month after injury. At that time, a diagnosis of arteriovenous fistula of the left midbrachial vessels was made. Compression of the artery proximal to the fistula caused a drop of 22 beats per minute of the pulse rate. On two occasions the patient had secondary hemorrhage from a small wound on the posterolateral aspect of the left arm. There was no nerve injury or cardiac enlargement. Operation was performed eight weeks after injury because of secondary hemorrhage and slight increase in the mass at the site of the fistula.

The fistula was dissected out after isolating the main vessels both proximally and distally. The vein was opened and a reparative endo-aneurysmorrhaphy was carried out, sacrificing the vein. An arteriogram, made at this time, showed the artery to be patent at site of repair. There was a good radial pulse postoperatively.

Case 7: This case was of interest because there was a typical machine-like murmur and thrill before operation, and yet no definite arterial communication could be found at operation. A 22-year-old Negro, male, received a pistol shot wound of the right side of the neck. The bullet entered just above the right sternoclavicular junction and the wound of exit passed through the scapula. For the first 12 hours the arm was cold and pulseless, then it became warm, and the pulse became normal. The patient was in a hospital in Corsica for one month, then returned to duty. Two weeks later he was readmitted because of numbness of the inner side of the arm, and a diagnosis of arteriovenous fistula of the subclavian vessels was made. The patient

was admitted to this hospital a few days later. The blood pressure of the two arms was equal, 110/70. The thrill and bruit were characteristic of an A-V fistula. However, the thrill and bruit could be obliterated by pressure proximal to the fistula *without* decreasing the volume of the right radial pulse or appreciably slowing the pulse rate. There were signs of compression of roots of C-7 and D-1, and impairment of pain and vibratory perceptions in the fourth and fifth fingers. There was no cardiac enlargement and the E. K. G. was normal.

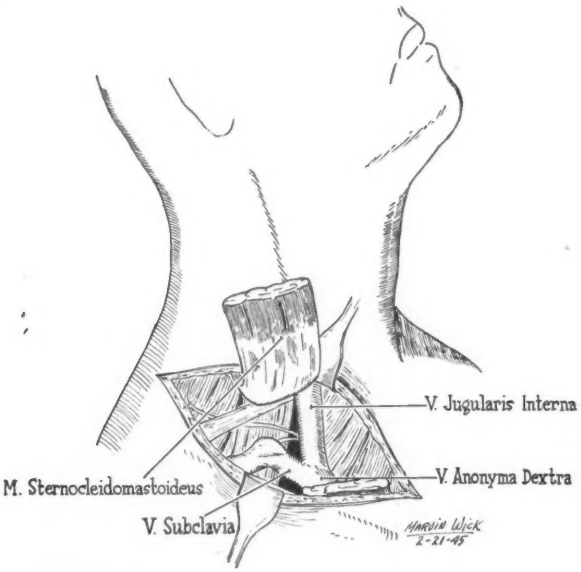

FIG. 4.—Drawing showing the angulation of the proximal portion of the subclavian vein.

At operation, the proximal portion of the subclavian vein was found to be angulated by scar tissue. At the apex of the angulation on the superior surface, the wall of the vein was quite thin, resembling a diverticulum (Fig. 4). Eddies of blood could be seen in the vein and the palpable thrill was quite marked. The thrill could be obliterated by temporary occlusion of either the proximal or distal segment of the subclavian vein. No arterial communication could be demonstrated. The involved segment, four centimeters in length, was excised. Postoperatively, the thrill and murmur were absent, and the patient was retuned to full duty. Careful examination of the specimen failed to reveal any communication.

COMMENT: It is hoped that further experience with the nonsuture anastomosis, as advocated by Blakemore, *et al.*, will lower the incidence of gangrene following sudden trauma to major arteries. This method would seem especially feasible in popliteal artery injuries, since injury to this artery was followed by gangrene in 86 per cent of the patients observed in this hospital.

Early operative intervention in traumatic ancurysms was necessary in

about 40 per cent of the patients seen. This includes several patients with injuries of only a few hours' duration. The systolic bruit and expansile pulsation disappeared spontaneously in three patients while awaiting evacuation. The operative incidence in this hospital was less than 30 per cent in patients with arteriovenous fistulae, excluding those patients who were returned to duty shortly after operation. We have not seen the spontaneous closure of any of the arteriovenous fistulae.

The temporary occlusion of vessels has been accomplished by using a broad cotton tape ligature tied down on a segment of small rubber tubing laid on top of the vessel—a method suggested by Holman.[6] Bulldog artery clamps not being available, we have also used a screw clamp to obtain partial, or complete, occlusion of the artery after encasing the vessel in a segment of tubing of suitable size. Manual compression by an assistant just proximal to the ligature decreases the tension so that the ligature can be tightened with less danger of rupturing the intima. All vessels are divided and the ends transfixed.

The subperiosteal resection of the medial half or two-thirds of the clavicle was done in two instances of axillary or subclavian artery injuries. This gives an excellent exposure and there is little apparent deformity or loss of function postoperatively. The periosteal bed is reconstructed at the time of closure. We have applied a figure-of-8 plaster yoke for three weeks postoperatively.

SUMMARY

In summary, we feel that persistent posttraumatic vasospasm should be eliminated either by paravertebral injections or preganglionic sympathectomy. Second, the incidence of gangrene following arterial ligation probably may be decreased by either early, repeated sympathetic injections or by early sympathectomy. Chronic arterial deficiency is common following ligation of the main artery of an extremity and in selected cases this may be greatly improved by sympathectomy. Finally, the results in 19 patients operated upon for traumatic aneurysm or arteriovenous fistulae are presented.

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CHRONIC HYPERTROPHIC ANTRUM GASTRITIS

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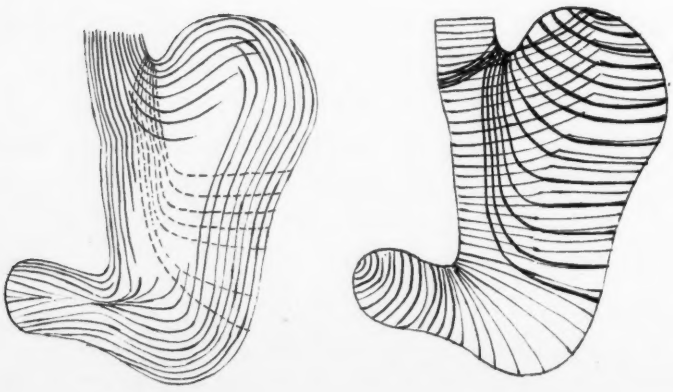
ANTRUM GASTRITIS is of the greatest clinical significance when one considers it as an ever-present differential diagnostic possibility. It has many clinical features in common with ulcer of the antral region. It has frequently deceiving similarity with a malignancy of the same region, roentgenologically and clinically.

The purpose of our study is to evaluate clinical and roentgenologic findings, to see how much reliance can be placed on them, and which cases should be sent to the gastroscopist as a last resort before surgery. We shall try to avoid, however, a shortcoming frequently found in the presentation of the subject—the presentation of the gastroscope rather than of gastritis. The instrument's value is not in doubt; but, as cystitis and nephritis can be diagnosed without the cystoscope, we should find the means of making the diagnosis of gastritis with simple, clinical methods.

The history is usually regarded as unreliable, but then it is an art to take a stomach history which is analytical and more than an indefinite report of sensations; yet, as experienced clinicians know, a good history with roentgenograms make a stomach diagnosis. In hypertrophic antrum gastritis the patient has no pain in general, but chooses "discomfort after eating, fullness, burning sensation" as terms of description. Yet, taking a sip of ice water or hot tea frequently elicits real pain, not localized but spread over the entire stomach.

At certain stages of the disease the stomach wall becomes tender to careful palpation or slight percussion. Antrum gastritis is frequently associated with cramp-like, painful sensations. Even before seeing his physician, the patient learns to avoid meat and prefers milk and a light diet. There is no seasonal let-up; the discomfort is usually persistent throughout the day and night for weeks and months. Only occasionally have we observed a case with severe clinical symptoms—weight loss, nausea, and vomiting. Bleeding, however, has often been noted. Hemorrhage leading to death has been observed. Benedict, in a recent publication, reports the incidence of hemorrhage as 19.7 per cent (43 to 213 cases). Peritonitis has occurred.

The next step in the examination should be aspiration of the stomach contents. This should not be an Ewald meal, but a fractionated aspiration after a cup of tea and a slice of buttered toast has been given. Such examination will show in chronic hypertrophic antrum gastritis a considerable incidence of hyperchlorhydria, definitely more frequent than in pangastritis. While such findings are helpful in the differential diagnosis of carcinoma, subacidity and anacidity are occasionally found, either due to increased mucous secretion or to the insufficiency of the secreting glands in later stages



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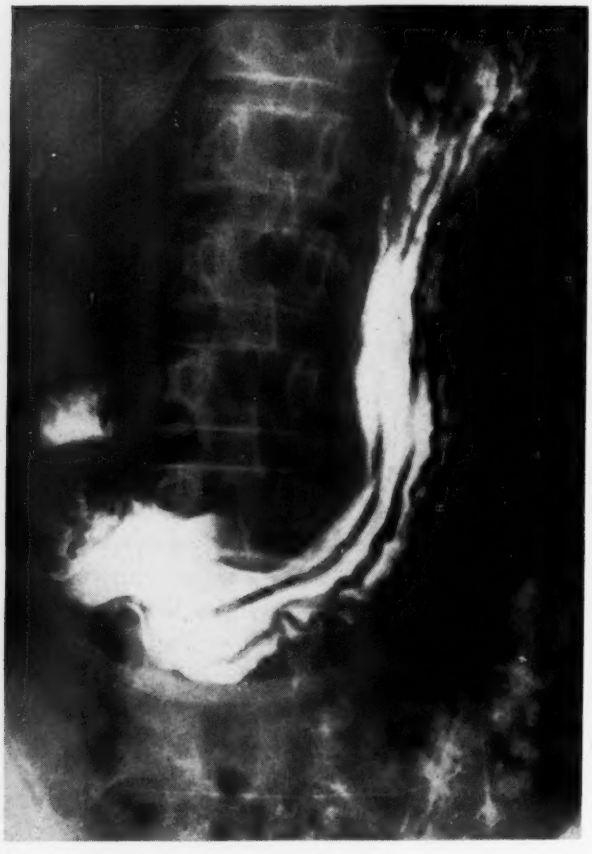

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FIG. 1.—A. Longitudinal.
B. Transverse and oblique musculature of the stomach, according to Forssell.
C. Mucosal folds of normal stomach under prevalence of longitudinal muscle contraction.

of the inflammation. In our opinion this does not greatly detract from the considerable value of the aspiration test, as other signs of inflammation might be found in the extract—increased white cells, red cells, and mucous secretion.

The next step in our examination is the roentgenologic examination of the stomach. The roentgenologist should have on hand all the clinical data thus far obtained. There are various distinctive signs of chronic hypertrophic antrum gastritis evident in roentgenograms. Some of them might well be

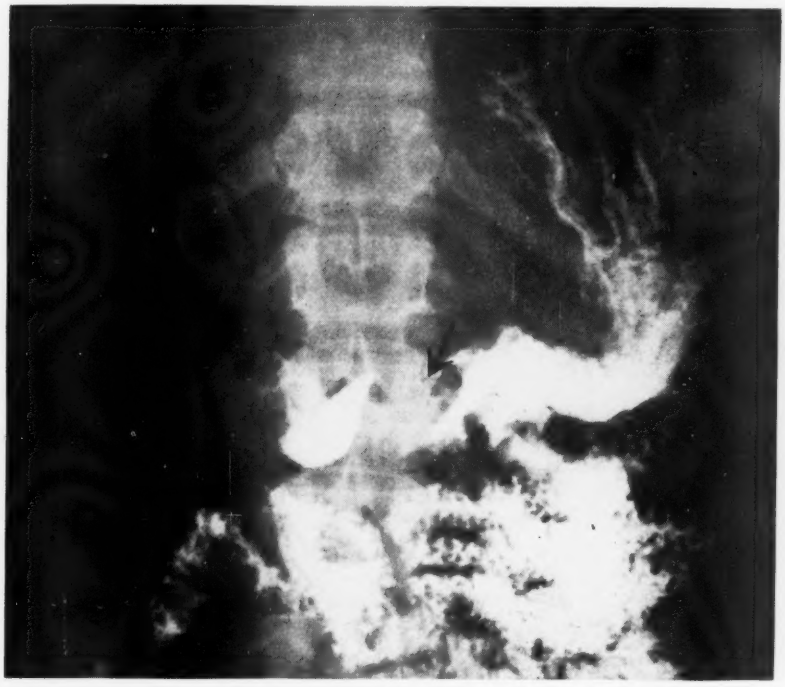

FIG. 2.—Case 1: Severe hypertrophic antrum gastritis in a girl 18 years of age. Large filling defects. Note borderfold not disrupted.

accessible for direct inspection, others are beyond the reach and limitation of the optical instrument.

The term "antrum gastritis" needs justification; in fact, the entire classification of gastritis demands a review. It should not be based solely on gastroscopic findings, but should have either an anatomic or physiologic basis. The classification of gastritis, according to Schindler, has great practical merits, but is not the only one possible. He differentiates: (1) Superficial gastritis; (2) atrophic gastritis; (3) hypertrophic gastritis; and (4) gastritis of the postoperative stomach. Such a classification shows gaps and incongruities; it suggests, for example, the question as to whether there is not a form of gastritis which, in contrast to a superficial gastritis, is one of the deeper layers; forms of interstitial gastritis; and stages of development of

the disease not accessible to direct inspection, at least not to a single examination.

Such cystic and nodular forms of gastritis have been described by pathologists as gastritis cystica superficialis and cystica profunda (Lubarsch). The question is important to the roentgenologist not as a contest between specialties, but as it might account for certain discrepancies between roentgenograms and negative gastroscopic findings.

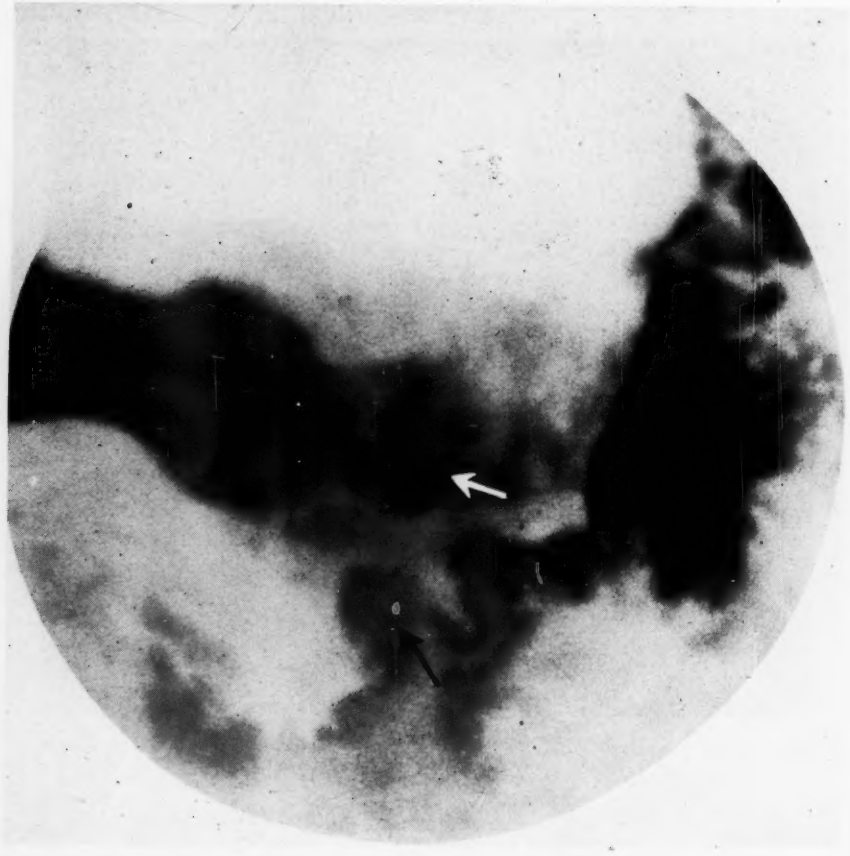

FIG. 3.—Case 1: Spot-film: Wall infiltration. Round filling defect. Antral narrowing.

Not all types of gastritis are exogenous (coming from the surface), such as is the case in alcoholic gastritis; otherwise, the considerable divergence between the advocates of the ulcer theory due to gastritis and those due to peptic-corrosive influence of the stomach secretion, would not be such a fundamental one. More frequently it is an elimination gastritis (Bourget) (coming from the depth after various infections). Gastritis has been observed accompanying influenza, cholecystitis, and even common colds; uremic gastritis is another type which comes from the depth to the surface.

Thus, the pathologist might choose a different principle of differentiation:

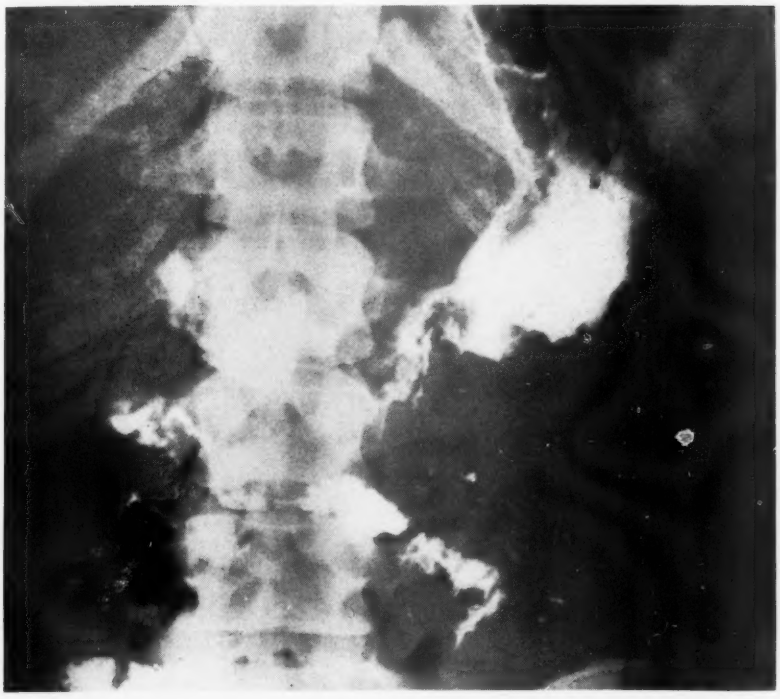

FIG. 4.—Case 1: Same case one week later. Progressive involvement.

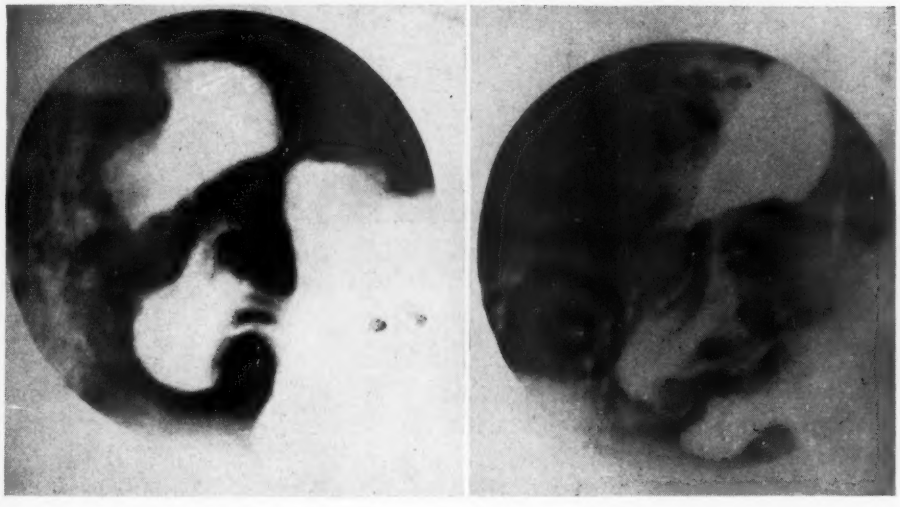

FIG. 5.—Case 1: Six months later peristalsis restored, only slight mucosal irregularity left.

(1) Topographic, whether diffuse or localized; (2) according to the type of inflammation; and (3) he might prefer, as would the clinician, to differentiate between gastritis of different etiology or propagation—endogenous and exogenous.

The term "antrum gastritis" could mean just such a topographic des-

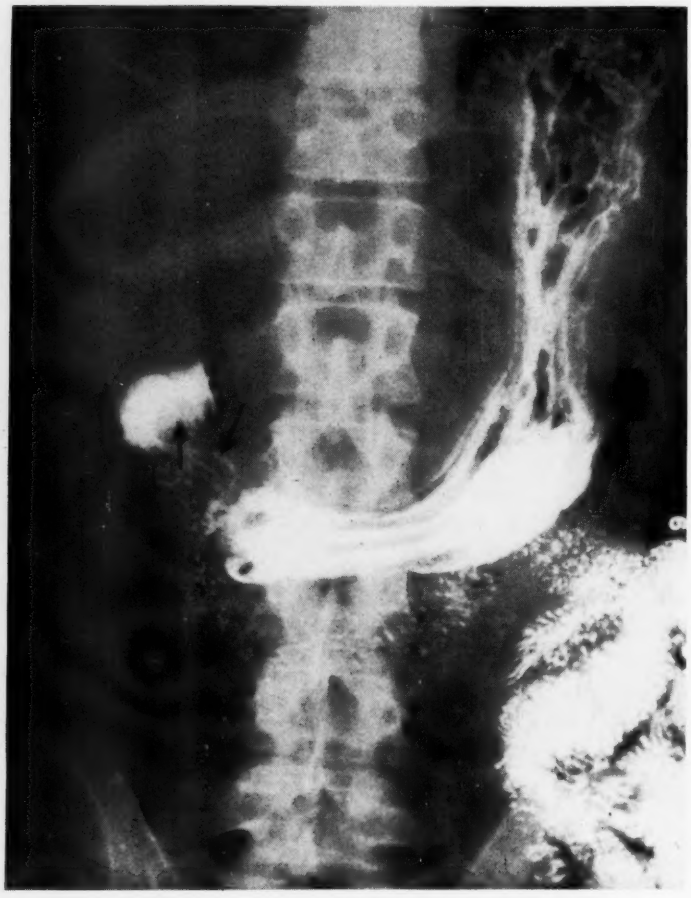

FIG. 6.—Case 2: Pylorus hypertrophy (Ross Golden) and Kirklin's sign: invagination of bulbar base present in hypertrophic antrum gastritis.

ignation, but as the term is not unanimously accepted, it requires clarification. As to the occurrence of gastritis limited to the antrum, veterinary medicine offers some interesting facts. Two authors, Bongart and Tantz, examined the stomach of 1,500 calves, and found at the age of four to five weeks an erosive gastritis in the second part of their stomach. When the calves were 12 to 14 weeks old, the authors found such erosive gastritis in 98 per cent; when they grew up and were one to one and one-half years old, no ulcers were found, but there were many star-shaped scars limited to the antral region. The microscopic examination proved them to be subacute or

chronic gastritis. The ulcers occur at the time when the calves are weaned, with transition from milk to raw fodder and the beginning of rumination.

This illustrates well that in addition to the chemical changes a mechanical factor plays an important rôle in the antral localization of gastritis. Experimentally induced gastritis in dogs is usually localized in the antrum.

In man we have frequently observed, roentgenologically, a pathologic involvement strictly limited to the antrum, and the anatomy of the antral region explains such localization. The antral systole creates considerable

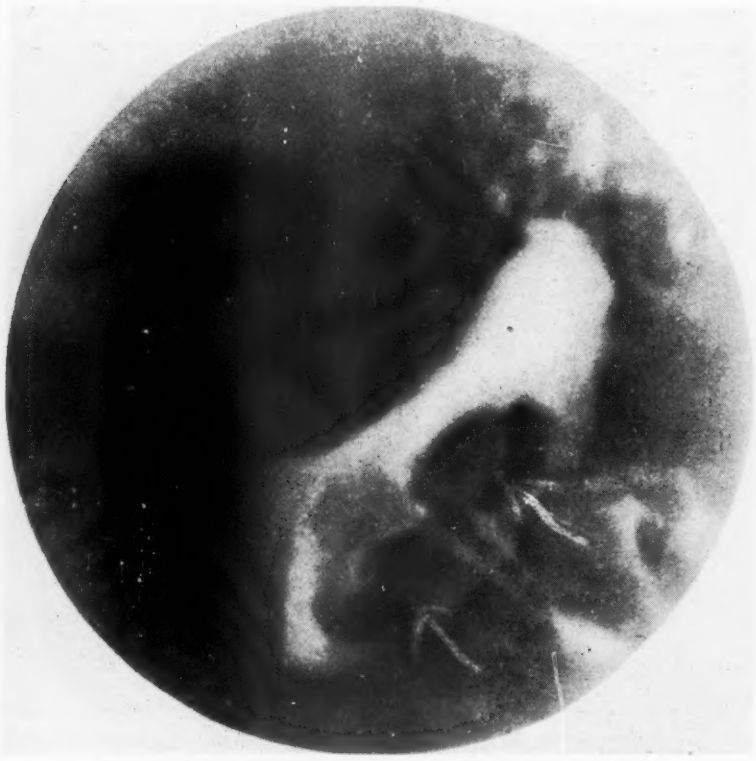

FIG. 7.—Case 2: Spot-film showing invagination of bulbar base.

pressure within the antral chamber. A spherical pressure chamber is created between the closed pylorus and the plica angularis; this contraction being soon arrested by spasm, and later on followed by muscular hypertrophy.

It is pointed out by Ross Golden that antrum gastritis is frequently accompanied by disturbance in motility, antral spasm, and prepyloric narrowing, and he quotes Serck Hanssen, who found gastritis "invariably present with hypertrophy of the pyloric muscle."

Thus, we have a roentgenologic sign helpful in the diagnosis of chronic hypertrophic gastritis: Visible pylorus hypertrophy or antral spasm in adults.

A second sign is a change in the mucosal pattern. The significance of the mucosal pattern for the recognition of early malignancy and ulcers cannot

be too highly emphasized. How much value does it have in the diagnosis of gastritis, in general, and of antrum gastritis, in particular?

The mucosal pattern of the stomach is neither something invariably frozen, like our skin lines, nor as variable as ocean waves. Gôsta Forssell, in his great work on the autoplasty of the mucosal pattern, has demonstrated and analyzed the great variation of the mucosal folds of the working stomach. In contrast to the working stomach, the fasting and empty stomach has a characteristic initial pattern. The region of the lesser curvature shows the system of the longitudinal folds, the fornix and the greater curvature, mainly

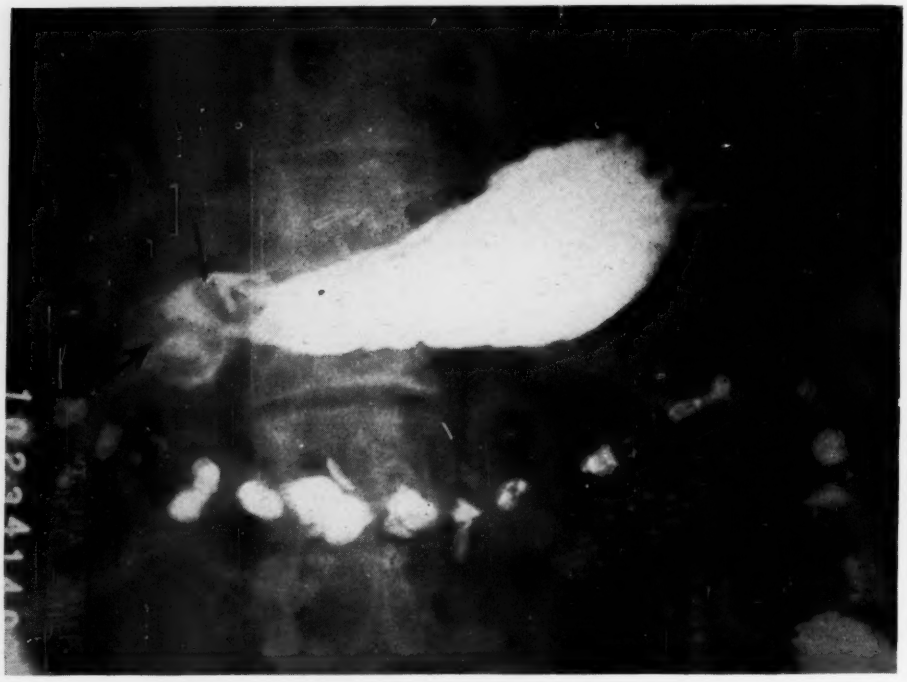

FIG. 8.—Case 3: Nodular hypertrophic gastritis. Confirmed by gastroscopy as three shallow nodular protrusions.

the system of the elliptic curved folds. In the region of the lower angle there is a quite characteristic, inverted Y-shaped fold—the plica angularis. Towards the antrum the folds are usually arranged in the pressure axis of the antrum. Several adjacent folds have usually the same direction. Upon the initial pattern is superimposed the working pattern, depending on functional requirements, hydrodynamics, and nervous impulses. After emptying, the stomach returns to its initial pattern.

How much this mucosal pattern conforms to the structure of the mucosal system is evident by comparison of the schematic drawing (according to Forssell) and a normal, regular roentgenogram taken of the fasting stomach (Fig. 1).

The constancy of the mucosal pattern of the empty stomach makes it possible for the roentgenologist to attach significance to any change in struc-

ture and direction of these folds, provided a proper and exacting technic is used. Only upon the changes in the inherent, basic design, and not upon the various autoplastic movements can roentgenologic diagnosis rely. In spite of certain variations the basic design in the antrum is sufficiently clear to recognize any major break and deviation. Roentgenology was able to demonstrate the convergence of mucosal folds around a duodenal ulcer, the torus form of the folds around a gastro-enterostomy, and even the Ascaris

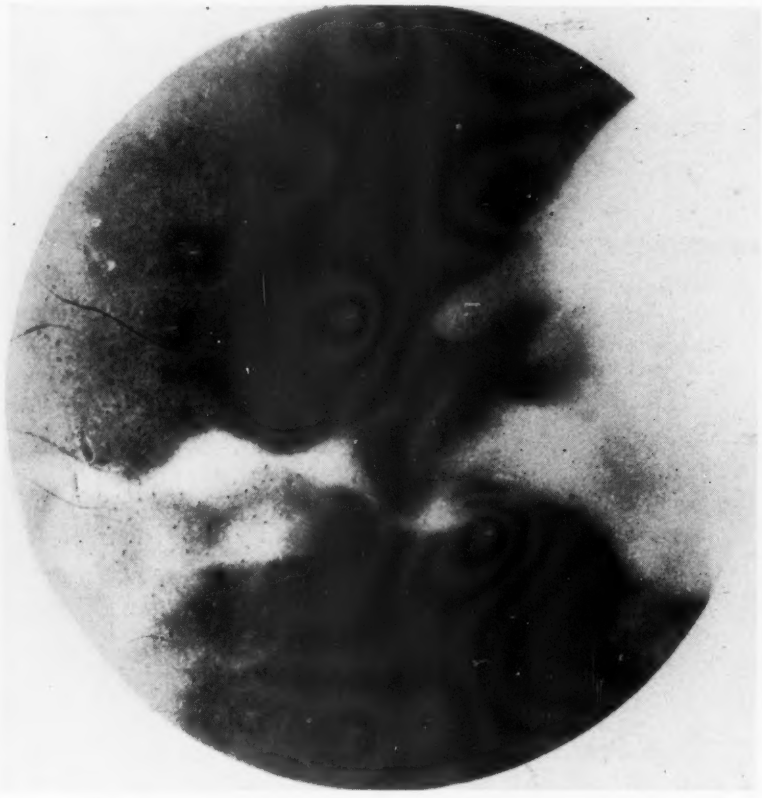

FIG. 9.—Case 3: Spot-film showing cobblestone appearance with preserved architecture.

worm in the duodenum. It should be relied upon to find a considerable swelling, stiffness, and deformity of the mucosal folds. This is not in contrast to Forssell's doctrine of the autoplastic, as he states that the anatomic structure at each side facilitates the formation of the pattern characteristic of that side.

Any break of the continuity of one or several folds; granular appearance of the mucosal folds; irregular translucencies; and abrupt changes in direction if found in an empty stomach are suggestive of pathology. The differential diagnosis between antrum carcinoma and chronic hypertrophic gastritis, is, admittedly, difficult; but frequently, as our examples demonstrate, the difficulties are not insurmountable.

The characteristic, grape-like appearance of polypi in the antrum gives the same roentgenologic and gastroscopic picture, whether they are of an inflammatory or neoplastic character. The small, wart-like elevation, not so rarely found in the antrum, and the single, elevated growth, flat or lobulated, are either the end-product of chronic hypertrophic gastritis or true adenoma, fibroadenoma, papilloma, or adenopapilloma. Only histologic study may decide it.

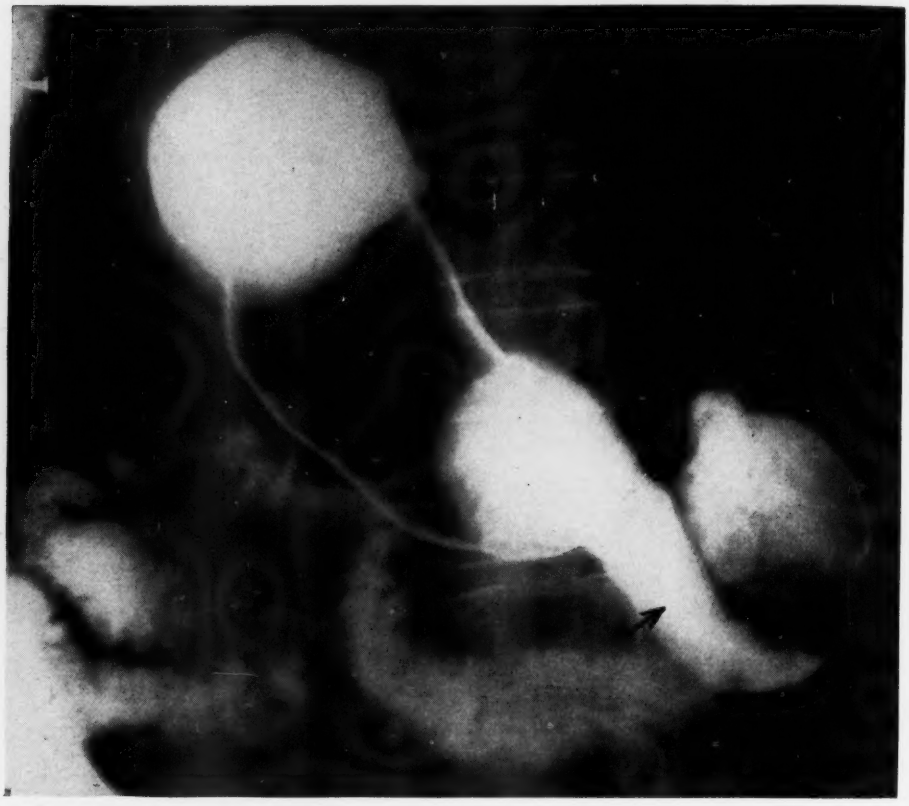

FIG. 10.—Case 4: Muscular hypertrophy of antrum with cellular infiltration. Antrum gastritis, not carcinoma.

Spriggs, in his exhaustive, well illustrated study of polypi, found that many of the cases diagnosed as polypi were, in fact, such of polypoid gastritis. Among his 19 cases of polypoid gastritis were seven in which the polypi developed after gastro-enterostomy or accompanied peptic ulcer. Brunn and Pearl found polypi in 85 per cent of their cases localized in the antrum, and in 35 per cent they were exclusively in the prepyloric region.

These wart-like elevations are, however, sometimes spread throughout the stomach and arise from an atrophic mucosa. (See Fig. 17.) The roentgenologic appearance in the antrum is that of a cobble stone street on a rainy day; the rivulets of barium passing around the cups. The malignancy has usually

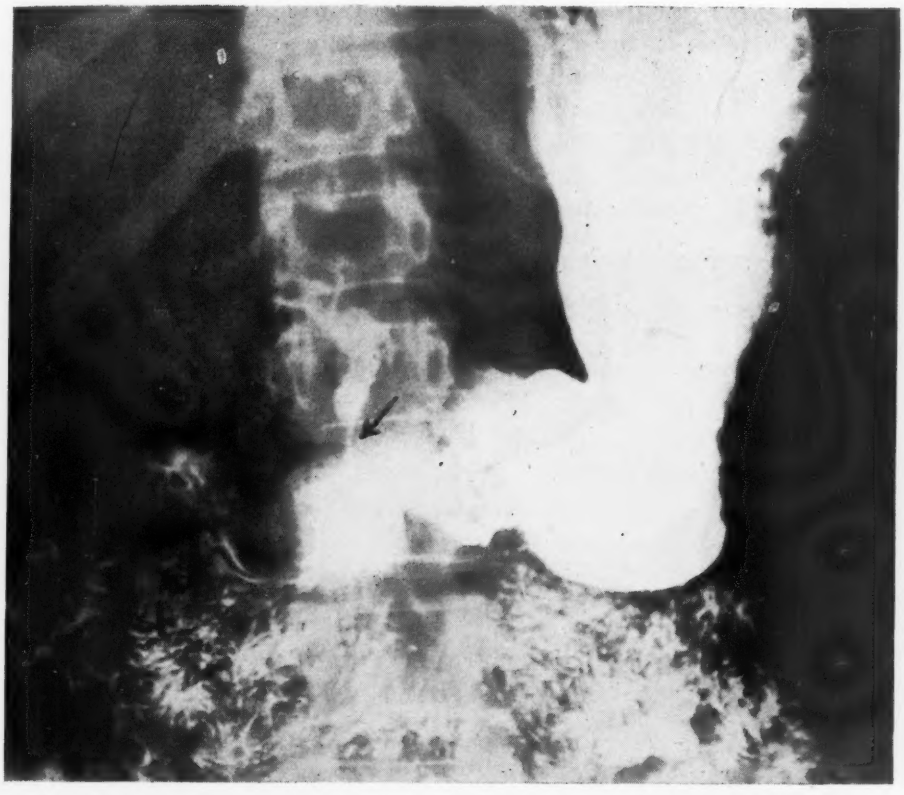

FIG. 11.—Case 5: Pylorus hypertrophy, cap deformity due to old ulcer scar. Antral defects in mucosal pattern. Antrum gastritis.

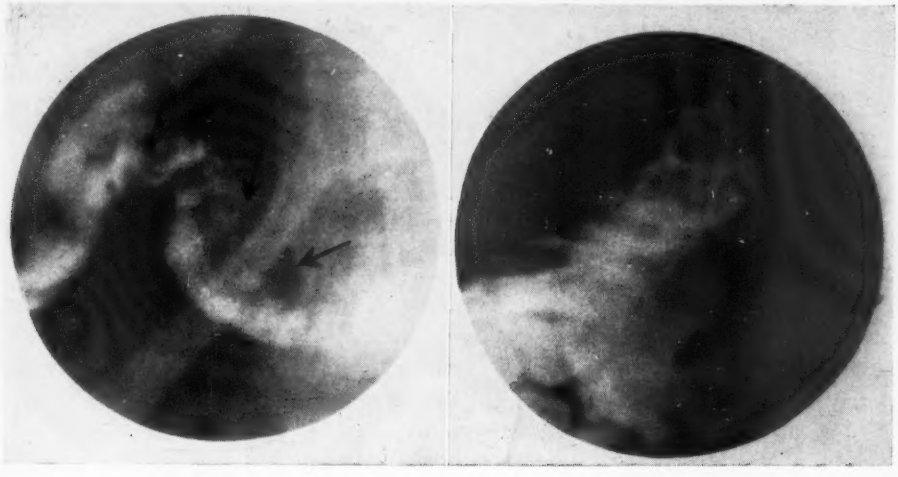

FIG. 12.—Case 5: Close-up view in spot-films.

a more vehement aspect, like a roadblock or a mine crater between the folds. Early opening of the pylorus with a deformed antrum is in favor of carcinoma; delayed opening with a similar deformity is in favor of antrum gastritis. As to differentiation between polypoid and hypertrophic gastritis on one hand, and antrum carcinoma on the other hand, the same general rule applies which Schindler gives for the gastroscopic differentiation: Regularity, granular appearance, and uniformity in size are in favor of gastritis; variability, and irregularity are suggestive of carcinoma.

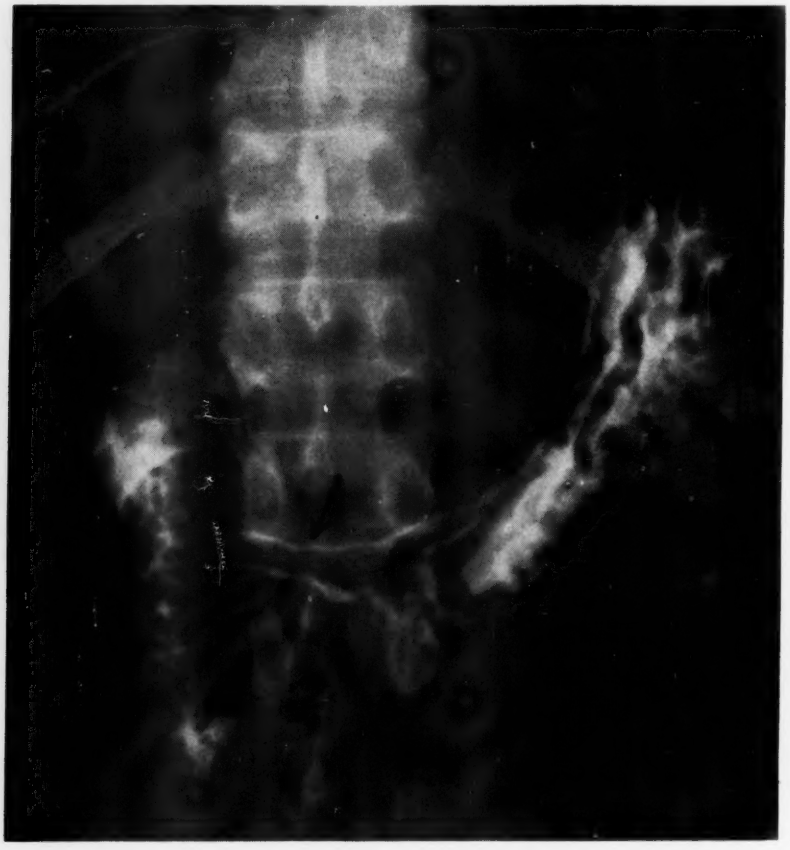

FIG. 13.—Case 6: Rigid hypertrophic antral folds. Note: Border fold not disrupted. Hypertrophic antrum gastritis. Complete regression after six months.

In cases which cannot be differentiated, we do not hesitate to advise operation. If we try to find the early stages of carcinoma, even an occasionally unnecessary operation is justified. The transillumination of the antrum with a strong light is helpful before opening the stomach.

Of other reliable signs of antrum gastritis of the hypertrophic type, a sign described by Kirklin and Harris was sometimes found to be helpful: The invagination of the bulbar base; a crescenting indentation of the bulbar base might mean mucosal prolapse. We are aware that one report from the

Presbyterian hospital, in Boston, mentions six cases where the sign was found unreliable in the differential diagnosis between carcinoma and hypertrophic gastritis. We found it of distinct value, and surgeons have frequently confirmed such pictures in the absence of carcinoma as due to thickened, sausage-shaped mucosal folds rising from the prepyloric region protruding into the bulb, causing stenosis.

Gastritis being often combined with duodenitis, which is visible roentgenologically, is another helpful sign. The sign of Steuer, however, the visibility of the gastric wall as a measure of the degree of wall infiltration, is of no help in antral gastritis and doubtful in pangastritis.

If the pathologic process reaches the Y-shaped border fold between the antrum and the stomach, plica mucosa-II (Elze), the close observation of that fold is especially indicated. A sudden break in its direction or continuity is in favor of malignancy; tortuosity, rigidity, and swelling speak for gastritis.

The most common type of chronic hypertrophic gastritis in the antrum is the one in which the mucosal folds are swollen, rigid, tortuous, but without any break in the structural relief. Too many warning posts have been erected in regard to the value of such findings, some of which, in our opinion, could safely be removed.

It is correct to keep in mind that mucosal folds rise and fall under the influence of drugs; that in cardiac failure, allergy, hay fever, and asthma folds might increase in diameter. Yet, we find in alcoholics, in ulcer patients, and in uremics a certain swelling and rigidity of the folds which we unhesitatingly diagnose as hypertrophic gastritis. The follow-up after proper management usually tells us how right we were. In doubtful cases we do not hesitate to advise gastroscopy.

A critical analysis of 938 cases of gastroscopic examinations has been published recently by R. I. F. Renshaw. There were 17.7 per cent with negative roentgenologic examinations where gastroscopy revealed gastritis; yet, gastroscopy was of no value in 19 per cent of the total cases.

In differentiation between a malignant and benign lesion both methods were approximately equal. Thus, even the combination of roentgenology, clinical study, and gastroscopy will still have a certain percentage of failures. In such cases it is our procedure to employ a period of dietary management, to follow the patient's blood count and weight curve, and then advise surgery, if the changes persist in a repeat examination. The close relation which exists between chronic hypertrophic gastritis, ulcer, and carcinoma, and the frequency of malignancy in the prepyloric region, justify surgical exploration in any doubtful case.

Case Reports

Case 1.—L. G., female, age 18, epigastric pains for three weeks, nausea, vomiting for three days, complete loss of appetite, tender epigastrium, free HCl 14, combined 16, blood in gastric contents 3 plus, bloodpicture: normal.

Roentgenologic Examination: Complete irregularity of mucosal pattern, antral wall infiltrated, stiff proliferative pressure zone.

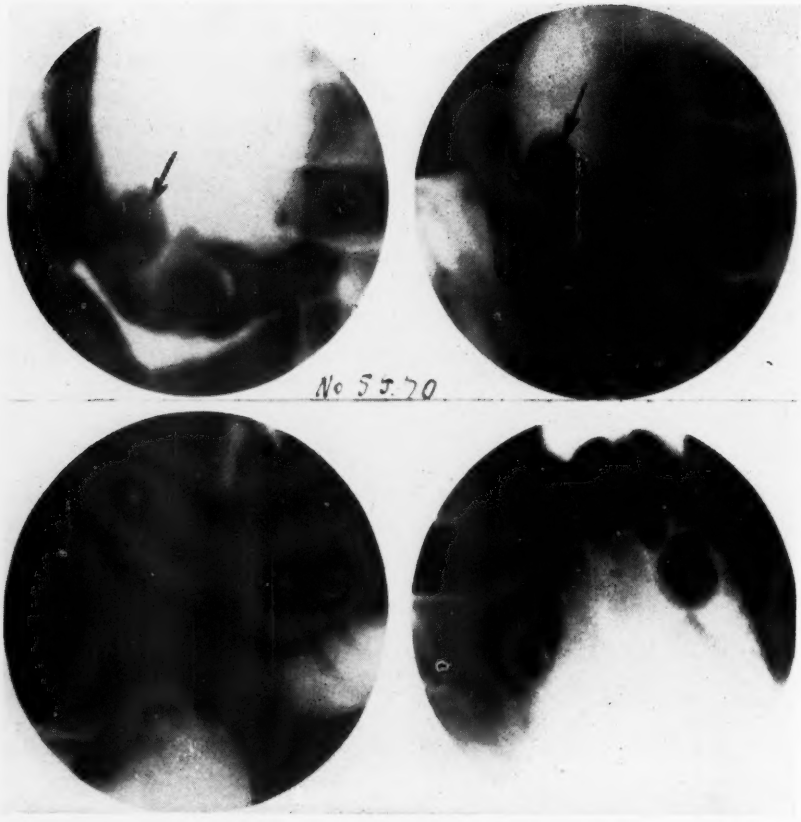

FIG. 14.—Case 7: Polypoid defects in the antrum in a generalized gastritis of the stomach. Roentgenologic diagnosis of hypertrophic antral gastritis, confirmed by gastroscopy and later, after resection, by histologic study.

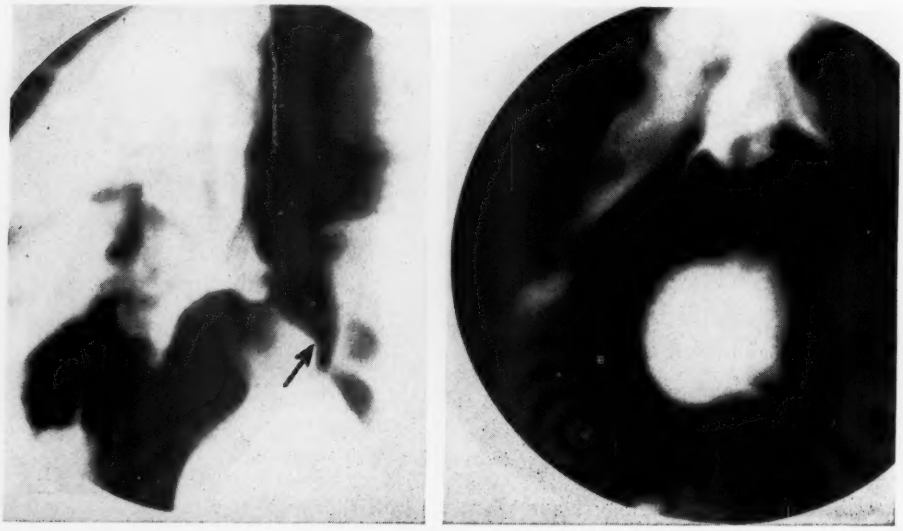

FIG. 15.—Case 7: Check-up one month later shows temporary regression, polypoid changes have disappeared, slight irregularity of medial prepyloric margin persists.

Diagnosis: Severe antrum and corpus gastritis. Gastroscopy advised, delayed because of bleeding and sickness of patient. Check-up films from month to month, progressive improvement and complete cure. Has led now, for five years, a healthy, married life. No stomach symptoms.

Case 2.—N. L., 46-year-old salesman, complaints of five months' duration, loss of 18 pounds in five months.

Clinical Diagnosis: Cirrhosis of liver.

Roentgenologic Examination: Esophageal varicosities, but also elevated, swollen, irregular folds of antral region. Kirklin's sign present: Folds invaginated into the duodenal base.

Gastroscopy: Hypertrophic gastritis and pigment spot in midstomach.

Case 3.—H. R., 32-year-old white male, sick for five weeks, joint pain, elevated temperature, malaise, backache for three weeks, epigastric distress of five weeks' duration, pain increases with food intake.

Roentgenologic Examination: Multiple nodular elevations in antrum region. Gastroscopic examination advised.

Diagnosis: Hypertrophic antrum gastritis.

Gastroscopy (Dr. Schindler): Definite pathologic changes are seen in the antrum wall of the distal portion of the antrum. Three shallow, protruding, dark red nodes were seen to lie in a soft noninfiltrated mucosa.

Gastroscopic Diagnosis: Nodular hypertrophic gastritis of the distal antrum with an hemorrhagic ulceration.

Case 4.—M. D., 64-year-old male, epigastric pains over six months, severe and cramp-like. Emesis four weeks ago, constipation, weakness, 20-pound weight loss.

Roentgenologic Diagnosis: Circular stenosis of antrum, regular outlines—carcinoma of antrum suspected. *Operation*—April 26, 1940: No pathology of stomach seen or palpated, resection of antrum, however, performed.

Histologic Study (Dr. I. Davidson) (Abstract). Marked antrum gastritis with cellular infiltration deep into the muscularis: *Muscular hypertrophy.* Arteriosclerosis.

This is a case of antrum gastritis in a patient 64 years of age. Muscular hypertrophy is an important roentgenologic sign of antrum gastritis, but differentiation from carcinoma of the antrum is not always possible.

Case 5.—B. F., 32-year-old female, blood in stools, epigastric distress, increasing constipation for the last few years.

Roentgenologic Diagnosis: Duodenal ulcer, besides abnormally heavy antral folds, suggestive of antrum gastritis.

Gastroscopy: Did not reveal any pathology.

Recent Roentgenologic Check-up: Three flowerbed-like elevations still present in the antrum. Unexplained by gastroscopy.

Case 6.—L. A., 50-year-old male, epigastric pain after meals, localized to midabdomen, no loss in weight, stool examination negative.

Roentgenologic Examination: Normal mucosal lining of the stomach down to the gastric angle. The border fold is preserved, important d/D sign. Only two thick folds can be followed through the antrum. Peristalsis over antrum sluggish but persistent. Severe duodenitis visible.

Diagnosis: Hypertrophic antrum gastritis. Patient refused gastroscopic check-up. After conservative treatment patient shows marked improvement; now free of symptoms; stomach normal after two years' observation.

Case 7.—(Courtesy of Doctor Staple). S. T., 46-year-old male, lived for four months only on milk, toast, and cottage cheese; cannot digest any meat, has diarrhea, foamy stools, gastric contents: 10/34 (15 min.), 31/45 (35 min.), 32/46 (50 min.), 6/43 (70 min.), much mucosa admixed.

Roentgenologic Examination.—September 22, 1941: Besides an ulcer niche at the

lesser curve of the stomach, the rugae of the antrum show several sharply outlined fillings defects. Peristalsis passes over this area; moderate pyloric stenosis.

Diagnosis: Antrum gastritis with polypoid fillings defects. Gastric ulcer.

Gastroscopy (Doctor Schindler): Definite mucosal changes were seen in the great curvature, depth-I., there the mucosa was dull, being cut into small polygons by many black creases and crevices. The folds were not affected. No ulcer was found in the usual ulcer area above the angulus, but the upper portions of the posterior wall showed, again, swellings and formation of nodes.

Gastroscopic Impression: (1) Localized hypertrophic gastritis. (2) Lues.

Roentgenologic Check-up.—October 22, 1941: Marked improvement; round defects in antrum have disappeared, but rugae are still thickened; ulcer niche as before.

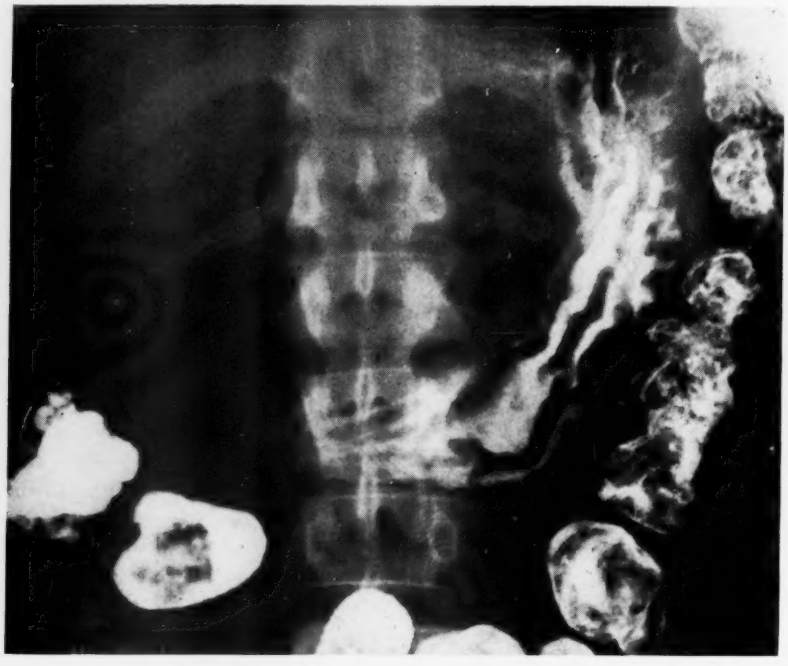

FIG. 16: Serum reaction: Acute swelling of mucosal folds occurred after blood transfusion, imitating hypertrophic gastritis.

Gastroscopic Check-up: Antrum and pylorus normal. A few nodules between the folds of the body were the only marks of the former severe hypertrophic gastritis.

Gastroscopic Impression: Remnants of hypertrophic gastritis.

Operation.—September 29, 1942: *Pathologic Findings:* Mucosa appears hypertrophic, rugae are distinct; about 10 cm. from pylorus is an area of induration, 3 cm. in diameter, the center of which presents an ulcer measuring 5 mm. The pylorus appears thickened, and there is moderate fibrosis of the duodenum beyond.

Microscopy: No evidence of a malignancy; chronic gastric ulcer; hypertrophic gastritis.

CONCLUSIONS

Chronic hypertrophic gastritis presents an ever challenging diagnostic problem, as it has clinical and roentgenologic features in common with ulcer and malignancy of the stomach.

The special problem here under discussion is that of chronic hypertrophic

antrum gastritis. There are certain anatomic and physiologic reasons for the frequent occurrence of such hypertrophic gastritis in the antral region.

The combination of clinical study, roentgenology, and gastroscopy permits a high degree of differentiation; yet, there is still a significant percentage of errors, as high as 20 per cent, and the search for simple diagnostic and roentgenologic signs should continue.

Roentgenologic signs of chronic hypertrophic gastritis are found more frequently in the antrum than elsewhere, or in gastritis of different type (atrophic). Some of these signs, such as Ross Golden's antrum hypertrophy,

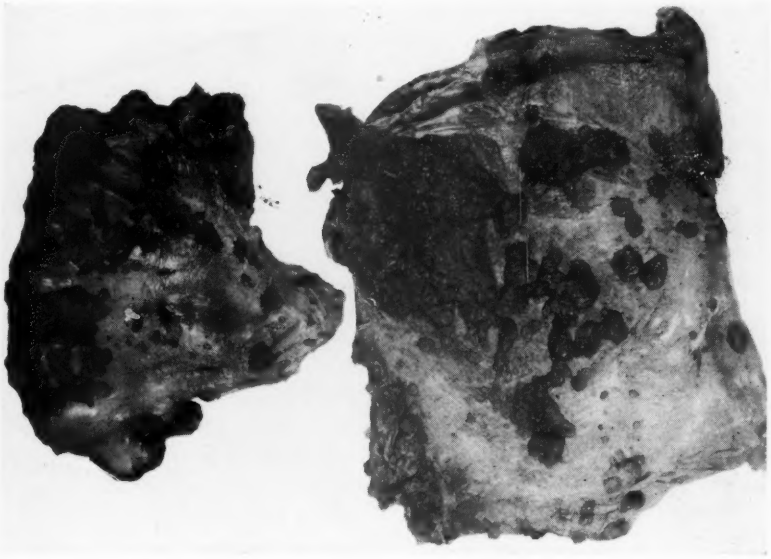

FIG. 17.—Residues of a polypoid hypertrophic gastritis in an atrophic mucosal field. Specimen.

Kirklin's base prolapse, and Steuer's wall symptom, are reviewed and briefly evaluated.

The constancy of the initial relief of the stomach in contrast to the variability of the working relief is stressed. Roentgenologic and differential diagnosis should be based upon the minute observation of the initial relief of the mucosa. Any vehement break in the contour and architecture of the mucosal pattern is suggestive of malignancy. Rugosity, stiffening, and widening of the folds, with preserved architecture, is highly suggestive of antrum gastritis, if supported by a history of pain and not contradicted by the clinical symptoms.

Early opening of the pylorus combined with antrum lesions is in favor of carcinoma; delayed opening is in favor of antrum gastritis. Polypous gastritis has a pathognomic roentgenologic appearance, but whether the process is of chronic inflammatory origin or neoplastic can only be decided by histologic examination.

Surgical intervention should not be delayed in doubtful cases beyond a limited period of medication and dietary management.

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EXPERIENCES WITH THE MILLER-ABBOTT TUBE*

A STATISTICAL STUDY OF 1000 CASES

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IN DECEMBER, 1937, the Miller-Abbott tube was introduced at the Presbyterian Hospital as a therapeutic and diagnostic instrument under the supervision of the surgical residents. Its use spread rapidly and there has been a daily average of six to ten, at times twelve, tubes in use on the Surgical, Gynecologic, Obstetric, Genito-urinary, Neurologic and Children's Services of the Presbyterian Center. To meet this demand, Dr. A. O. Whipple (Director of Surgery), in 1939, assigned the entire supervision of tubes and records of their use to a graduate nurse. She fills out a mimeographed sheet for each tube case. This information is transcribed to punch cards in the Record Room. Approximately 1,100 such records are now completed, and this paper is a statistical review of 1,000 of these cases.

I have personally reviewed 500 of these charts and found the results as recorded in close agreement with those of this entire statistical study.

The Miller-Abbott tube has made the small intestine accessible for physiologic observation, diagnosis and therapy.

Used prophylactically before operation, the tube has been successful in decreasing the incidence and clinical severity of postoperative ileus associated with operations upon the small intestines, large herniae, resections of the colon, and in the presence of peritonitis.

In right colectomy its advantages have been so striking that its rather universal use has permitted a single-stage resection to displace the formerly considered more conservative method of two-stage resection, with enterostomy.

Whereas, it has also been useful in deflating the small intestine following resections of the left colon and rectum, it has not been as effective in these cases in deflating the remaining colon and, thereby, obviating the necessity of a proximal colostomy.

Postoperatively, it has deflated the stomach, but it should not be used primarily for this purpose. Its greatest function is deflation of the small intestine.

This tube has been a means of preoperatively diagnosing the presence and site of small intestinal tumors, foreign bodies, adhesions, kinks, bands, bleeding sites and malformations.

At operation, it has served as a guide to the site of obstruction. It has also permitted the small intestine to be, so to speak, pleated on the tube, thereby, facilitating the retraction of the small intestine from the operative area.

*Read before The New York Surgical Society, March 14, 1945.

Postoperatively, it has maintained small intestinal deflation, which has improved the circulation in the intestinal wall, thereby, permitting more normal peristalsis and absorption, has aided wound healing of an anastomotic site, decreased the incident of fistulae and, in the majority of cases, has been an adequate substitute for enterostomy.

It has been a means of administering fluids, proteins, sodium chloride, glucose and medications—but this function, in our hands, has been of secondary importance.

It has been a most important factor in reducing the mortality of mechanical and paralytic ileus. Leigh and Diffendorf,[1] in 1939, reported a mortality of 6.5 per cent in 182 cases of acute mechanical ileus from the Presbyterian Hospital which had been treated with the Miller-Abbott tube, with and without operation. From the same institution Smith and vanBeuren[2] reported a mortality of 18.4 per cent in 103 cases of acute mechanical ileus which had been operated upon without the use of the Miller-Abbott tube.

The cooperation of the Roentgenologic Department is necessary to obtain the maximum information and results from the use of this tube. Fluoroscopic observations and roentgenograms, with and without a barium mixture *via* the tube, have given diagnostic information which otherwise could only have been obtained by celiotomy. Barium may stop locally, pass on, or be regurgitated into proximal loops. It may advance but the tube may not, thus, indicating the degree of obstruction. The tube may be retarded by an obstructing kink, band, adherent loop, foreign body, tumor, inflammation or edema of the wall or extrinsic pressure. Multiple partial obstructions may be demonstrated.

The most important clinical fact connected with the use of the Miller-Abbott tube is the assurance that intestine with compromised blood supply is not present while the tube is being used. In mechanical ileus the pain is colicky, cramp-like, with free intervals. In ileus with compromised blood supply the pain is continuous. In mechanical ileus the pulse rate, temperature and white blood cell count are, as a rule, not elevated. In strangulation ileus the pulse rate and temperature increase with the duration of strangulation, and there is usually a corresponding rise in the white blood cell count. In mechanical ileus tenderness and muscle spasm is not noticeable. In strangulation ileus tenderness is usually marked over the area of compromised intestine. The differential diagnosis between these two types of ileus is of the greatest importance—for if strangulation ileus is present, delay in operation is not warranted.

The proper use of the Miller-Abbott tube may be life-saving. It needs one specially trained in its passage, familiar with its indications, and capable of observing it often enough to recognize the accomplishment of its functions, or detect indications of its failure and quickly test and adjust it. In contrast to this, the occasional and haphazard observations by untrained house and attending staffs has, unwarrantedly, more and more, discredited the use of

this tube and caused it to be discarded as ineffective, and this, according to our experience, is unjustified.

TECHNIC OF INTRODUCTION

The tube should be used as follows: The nasal passage is inspected for adequate patency; it, together with the pharynx, is anesthetized (2% butyn or Cocaine); the balloon is folded umbrella-like back over the tube, well lubricated, and with the patient partially upright, it is passed through the nostril into the stomach to the 60-cm. mark. Sipping water aids its more rapid passage. The stomach is deflated of gas and fluid with a syringe, then two to three cubic centimeters of metallic mercury is introduced into the deflated balloon. Ten to 15 cc. of air is necessary to push the mercury into the balloon. This air is then aspirated. The patient then lies on his right side and the tube is further introduced to the 75-cm. mark. The tip may be placed at the pylorus, under the fluoroscope, with the tube lying along the greater curvature. It is held lightly by adhesive to the nose (*not* the cheek), for if the slack lies along the greater curvature, there is sufficient tubing in the stomach to allow the tip to pass through the pylorus without introducing more tube, which may coil.

The progress of the tube can be noted by fluoroscopy and recorded by roentgenograms. The possibility of damage to the patient's skin by overexposure to the roentgen ray must be borne in mind.

Its passage through the pylorus is facilitated by manipulation under the fluoroscope. This requires experienced observations in a minimum of time. If fluoroscopic observations are not used, clear fluids may be taken in order to propel the tube to the pylorus. Once through the pylorus, the nasal tape is removed and the balloon is inflated with 10 to 20 cc. of air, and allowed to progress of its own accord. Peristalsis carries the inflated balloon downward, causing a noticeable tug at the nose. In paralytic ileus the tube may not advance rapidly, and requires more attention by suction with a syringe to deflate atonic coils of intestine. When the tube is not carried on by vigorous peristalsis it may be advanced by the patient or nurse two inches each two to four hours, but no faster, lest it coil in the stomach. This much slack in the stomach may allow the tube to advance with less vigorous peristalsis, but the possibility of it coiling should be kept in mind and checked by fluoroscopy. Patients are so grateful for the relief after gastric deflation that they usually cooperate with subsequent tube care. The position of the tube tip can be determined (1) by aspiration of bile or jejunal contents; (2) by the time required for fluid by mouth to be aspirated; (3) by lack of resistance to inflation of the balloon if it remains in the stomach; and (4) by roentgenograms or fluoroscopy. We do not hesitate to transport sick cases to fluoroscopy for observation and manipulation of the tube.

The time, amount and character of the aspirated fluid should be charted. The hematocrit, plasma protein and blood chloride should be followed. Sodium chloride replacement should be approximately five grams for each

TABLE I

AGE INCIDENCE

0–9	2
10–19	32
20–29	96
30–39	192
40–49	214
50–59	214
60–69	164
70–79	72
80–89	12
90, over	2

SEX INCIDENCE

Male	436	Female	564

TABLE II

TYPE OF CASE

Small bowel ileus:	
Paralytic	308
Mechanical	299
Due to vascular lesion	2
Large bowel obstruction	100
Small bowel prophylactic:	
Preoperative	151
Postoperative	84
Large bowel prophylactic:	
Preoperative	108
Postoperative	15
Diagnostic intubation	153
Therapeutic intubation	61

TABLE III

SPECIFIC CAUSE OF ILEUS

Peritonitis	154
Pneumonia	15
Disease of C. N. S.	3
Gangrene—strangulation	20
Bands and adhesions:	
Early postoperative	47
Late postoperative	127
Without previous operation	16
Diverticulitis	19
Volvulus	17
Intussusception	3
External hernia	32
Neoplasm	19
Regional enteritis	20
Postoperative distention	183
Hypoproteinemia	30

TABLE IV

OTHER CAUSES FOR INTUBATION

Peritonitis	35
Resection	142
For site of obstruction	142
For site of bleeding	11
Fhysiologic study	1
Peeding	9

TABLE V

SITE OF OBSTRUCTION IN MECHANICAL ILEUS

Not disclosed	228
Duodenum	10
Jejunum	82
Ileum	321
Cecum	59
Ascending colon	42
Transverse colon	48
Descending colon	73
Sigmoid	8
Rectum	36

TABLE VI

DURATION OF SYMPTOMS BEFORE INTUBATION

0–11 hours	52
12–23 hours	61
24–47 hours	94
48–71 hours	88
3–4 days	122
5–6 days	53
1 week	49
2 weeks	39
3 weeks, or more	242

TABLE VII

DISTENTION BEFORE INTUBATION

None	243
Slight	154
Moderate	316
Marked	197
Dilated loops (by x-ray)	412
Fluid levels (by x-ray)	247

TABLE VIII

TIME REQUIRED TO CONTROL SYMPTOMS

Not controlled	182
0–5 hours	112
6–11 hours	63
12–23 hours	163
24–47 hours	122
2–4 days	135
5 days, or more	30

TABLE IX

TIME REQUIRED TO PASS PYLORUS

Incomplete	33
Failure	221
1–2 hours	91
3–5 hours	95
6–11 hours	63
12–23 hours	220
24–47 hours	129
2–4 days	118
5 days or more	20

TABLE X

TIME REQUIRED TO REACH CECUM OR OBSTRUCTION

Not reached	361
0–23 hours	34
24–47 hours	52
2 days	5_0
3–4 days	10_5
5–6 days	7_7
1 week, or more	3_1
Not necessary	26_8

TABLE XI

TOTAL TIME OF INTUBATION

0–47 hours	54
2–5 days	434
6–9 days	310
10–13 days	103
14–20 days	68
3 weeks, or more	15

TABLE XII

CAUSE OF FAILURE TO PASS PYLORUS

Not possible	80
Not necessary	109
Poor management	17
Hiccough	4
Pylorospasm	1
No x-ray guidance	604

Table XIII

ROENTGENOLOGIC GUIDANCE

Not used	339
Used	543
Placed at pylorus at x-ray, on first attempt	239
on second attempt	43
on third, or more attempts	24
Manipulated through pylorus at x-ray	256
Passed spontaneously after placing at pylorus	144
Obstruction demonstrated by failure to advance	106
by barium	100
Patency of bowel demonstrated by tube at cecum	92
by barium	93
Barium studies—paralytic intestine	14
dilated loop	38
constriction	75
Angulation	62
filling defect	8
other—specify	50
X-ray diagnosis confirmed	85

Table XIV

TIME OF EXPOSURE AT FLUOROSCOPY

1–4 minutes	71
5–9 minutes	117
10–14 minutes	92
15–19 minutes	62
20–29 minutes	44
30 minutes, or more	38

Table XV

OPERATION

None	444
Exploratory	73
Division of adhesions	105
Resection	155
Enterostomy	24
Cecostomy	28
Colostomy	27
Enterocolostomy	6

Table XVI

USE OF TUBE

Removed before operation	52
Used as guide during operation	37
Left in place postoperatively	295
Allowed to advance postoperatively	277
Suction continued:	
0–23 hours	44
24–47 hours	75
2–4 days	300
5–6 days	170
1 week, or more	143

Table XVII

TREATMENTS USED WITH INTUBATION

High protein therapy	81
Vitamin therapy	53
Poultice, rectal tube	284
Rectal	605
Pitressin	71
Prostigmine	124
Morphine	446
Chemotherapy	315
Gastric lavage	52
Gastric suction	128

Table XVIII

FLUIDS AND TRANSFUSION

Parenteral fluids required:	
1 day	50
2 days	96
3–4 days	282
5–6 days	205
7–13 days	183
2 weeks, or more	48
Transfusion:	
whole blood	345
split protein	2
plasma	48
Fluid balance followed by hematocrit	676

Table XIX

MILLER-ABBOTT TUBE

Comparison of Intestinal Drainage with Fluid Intake (Oral)	1st Day	2nd Day	3rd Day	4th Day	5th Day	6th Day	7th Day
Drainage exceeded intake:							
by more than 2,000 cc.	21	21	20	9	6	1	4
by 1,500—2,000 cc.	15	22	17	10	4	1	1
by 1,000—1,500 cc.	38	34	16	17	5	5	3
by 500—1,000 cc.	69	64	59	44	11	12	4
by 0– 500 cc.	223	156	90	55	37	18	10
Intake exceeded drainage:							
by 0– 500 cc.	188	168	158	103	65	41	17
by 500–1,000 cc.	99	138	135	100	71	48	21
by 1,000–1,500 cc.	62	95	96	100	72	39	28
by 1,500–2,000 cc.	35	44	46	38	32	36	25
by more than 2,000 cc.	26	33	38	41	39	31	23
Equal	71	57	43	28	11	7	7

TABLE XX

COMPLICATIONS OCCURRING WITH TUBE *in situ*

None	834
Trivial:	
Sore throat	129
Sore nose	20
Earache	11
Eye pain	1
Other trivial complications	2
Serious:	
Ulceration	2
Perforation	1
Hemorrhage	3
Other serious complications	5

TABLE XXI

SYMPTOMS RECURRING AFTER DEFLATION

Early	140
Late	36
None	668
Gastric distention	132
Small intestine distention	119
Partial withdrawal of tube required	54
Second deflation required	68
Operation required:	
Immediate secondary	27
Delayed primary	15

TABLE XXII

ACCIDENTS IN INTUBATION

None	834
Requiring replacement	56
Not requiring replacement	11
Knotting of tube	10
Leaking balloon	15
Broken balloon	7
Fluid in balloon	13

TABLE XXIII

CASES WITH MORE THAN ONE INTUBATION

2nd intubation	79
3rd intubation	10
4th intubation	5
5th intubation	2
6th intubation	1
Use of mercury:	64
Used 2 cc.	50
Used 3 cc.	14

TABLE XXIV

SUMMARY

Intubation:	
Successful	751
Unsuccessful	219
Incomplete records	30
	1,000
Result:	
Improved	786
Unimproved	80
Died	100
Incomplete records	34
	1,000

liter of intestinal contents aspirated. Continuous aspiration with the tube tip in the jejunum returns one-half to two-thirds of the fluid taken by mouth, whereas, about one-third returns with the tip in the ileum.

If redistention along the tube proximal to the tip occurs, the tip should be withdrawn until it lies in the upper jejunum and again be allowed to descend and deflate the redistended loops. The stomach may become distended while the tube tip is deflating the jejunum, in which case gastric lavage with a Levine tube may be necessary. The Miller-Abbott tube should be clamped and left *in situ* for 24 hours, and not removed until the obstruction is relieved or passed; or the tip has reached the cecum; or the character of the aspirated fluid becomes normal intestinal contents, and the Roentgenologic Department agrees that its diagnostic possibilities have been exhausted.

The tables are a statistical study of 1,000 cases at the Presbyterian Hospital, New York City, from December, 1937, to February 21, 1945.

The serious complications cannot be attributed entirely to intubation. Both cases of ulceration occurred in patients with uremia. In each instance a superficial ulceration was found in the region of the aryepiglottic folds. Perforation occurred in the ileum of a case when, at operation, the adhered ileum was separated from an adherent area which obstructed the intestine. The tube was used as a guide to the obstructed site where the tip had been arrested. The presence of the tube tip was not an apparent factor in the perforation in this case.

Hemorrhage occurred following the introduction of the tube in an elderly patient, with subsequently proven esophageal varices. The presence of cirrhosis of the liver had not been appreciated prior to the passage of the tube. The other two cases of hemorrhage were in patients with an old history of duodenal ulcer. Melena occurred while the tube was *in situ*. The hemorrhage was not severe, and its origin was never proven.

The other five instances of serious complications could not be directly attributed to the tube. The most serious of these was the onset of bilateral vocal cord paralysis in an elderly patient, necessitating a tracheotomy. The tube had been *in situ* for 12 hours before the sudden onset of this complication. Although not proven, the etiology here was suspected to have been a sudden intracranial vascular lesion. The other complications were wound disruptions, postoperative pneumonia, and quite marked dehydration, necessitating parenteral fluids in a patient who would not take fluids by mouth with the tube *in situ*.

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THE SURGICAL SIGNIFICANCE OF AN ANOMALOUS CHOLECYSTOHEPATIC DUCT

CASE REPORTS

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THE SURGICAL SIGNIFICANCE of variations in the anatomy of the biliary system has been the subject of numerous contributions. Concerning the incidence of abnormalities, Flint[1] has shown in a study of 200 consecutive autopsies that there were only 69 cases in which the anatomic pattern of the extrahepatic biliary system (and its associated vascular tree) conformed to the textbook description. An anomaly to which attention has been directed, only infrequently, and which is the subject of this paper is an accessory hepatic duct draining a varying segment of hepatic parenchyma directly into the gallbladder. This biliary pathway, which we shall term the "cholecystohepatic" duct, is found regularly in fish, reptiles and birds (Owen[2]). According to Quain,[3] it is not an unusual finding in some mammals.

Both the occurrence and the surgical significance of the duct in man can be appreciated by a consideration of the embryology of the biliary system.[4] The latter begins with the appearance of the hepatic diverticulum, a saccular pouch from the ventral foregut. The distal end of the pouch is the source of solid cellular strands of tissue from which the glandular portion of the liver is formed. The cellular cords assume, gradually, the pattern of adult hepatic architecture chiefly as the result of invading blood vessels and biliary capillaries. Parallel to, but separate from, the foregoing development is the evolution of the extrahepatic biliary system from a hollow pouch immediately proximal to the developing liver. The distal portion of the pouch becomes the hepatic duct, which sprouts numerous small ducts that enter the substance of the liver. The proximal segment of the pouch (derived from the primitive duodenum) becomes the choledochus. At a variable point between the proximal and distal portions of the pouch is the special offshoot from which the gallbladder and cystic duct are derived. Thus, the gallbladder and hepatic duct are essentially distal segments of the same diverticulum. Presumably because of some error of development small ducts may sprout from the gallbladder as well as the hepatic duct to drain glandular buds of liver tissue. The latter, merging with the fetal liver would, nevertheless, continue to drain into the gallbladder. This appears to be the manner of development of the cholecystohepatic duct which, of necessity, drains a segment of the liver.

It is quite impossible to determine from the literature the incidence of an aberrant duct in man. There are individual case reports, such as an early

one (1913) by Kehr,[5] and one by Schnacher[6] in a review of extrahepatic biliary anomalies. Eisendrath,[7] in a basic paper on anatomic variations of the biliary tree, mentions no instance. The anomaly was not noted apparently in Flint's[1] study, or in 194 dissections by Luge.[8] On the other hand, bile capillaries and often larger bile ducts were found in the gallbladder bed in 15 to 25 per cent of cholecystectomies.[9] Mentzer[10] believes that the duct can be frequently overlooked at operation because of its (usually) insignificant caliber and, also, the reduction of biliary secretion under anesthesia.

Although the incidence of cholecystohepatic ducts remains doubtful or unknown, and the question of postcholecystectomy biliary discharge resulting from severance of such ducts remains open, there can be no doubt as to the hazard of an overlooked severance of a duct of substantial proportions. The excessive loss of bile by external drainage or the likelihood of bile peritonitis from internal drainage need only be mentioned. The obvious management of a small duct which is encountered at cholecystectomy is ligation or sealing by electrocoagulation. When, however, the diameter is large enough to indicate that the duct drains a substantial section of hepatic parenchyma there can be some doubt as to procedure. Thus, Flint[1] believes that intrahepatic collateral biliary circulation is sufficient to prevent liver damage following ligation. He concedes, however, that, in the case of an already damaged liver resulting from prolonged obstruction of the common duct, further insult to a substantial segment of liver by ligation of a large aberrant duct might lead to death. On the other hand, external drainage from an aberrant duct invites local complications, excessive biliary deprivation, and prolonged morbidity. A number of years ago we encountered an aberrant cholecystohepatic duct at cholecystectomy, and employed external drainage. Another case was seen recently, and this time the duct was treated by ligation.

CASE REPORTS

Case 1.—(A. R. 431484): A 58-year-old female entered the Mt. Sinai Hospital with a four-day history of right upper quadrant abdominal pain and jaundice. Abnormally dark urine was noted but the color of the stools was not observed. The patient had not suffered previous episodes of abdominal pain or icterus, but had had dyspeptic symptoms for a long time. Aside from frequent attacks of bronchial asthma, her past history was not noteworthy.

Physical Examination: The patient was obese and moderately icteric. Her abdomen was distended. There was marked tenderness and spasticity in the right upper quadrant where an orange-sized, tender, ballotable mass was palpable, moving with respiration. There was slight fever. *Preoperative Diagnosis:* Common duct obstruction and a distended gallbladder. After preliminary preparation with intravenous glucose solution operation was undertaken.

Operation: A right upper abdominal incision disclosed extensive pericholecystic adhesions arising from an enlarged, tensely distended gallbladder. The foramen of Winslow was obliterated by adhesions. Indeed, the diseased gallbladder was quite completely walled-off from the free peritoneal cavity. Cholecystectomy was performed, subserosally, from the fundus downwards. The cystic duct was about three millimeters in diameter, and, after its severance, several cubic centimeters of dark, very viscid bile containing numerous fine crystalline particles, but no stones, escaped from its stump.

Thereafter only thin yellow bile welled-up from the common duct. Exploration of the common duct by palpation and by probe revealed a duct of normal diameter containing no calculi. Since compression and distortion of the duct system by the gallbladder mass appeared capable of producing the icterus of a few days' duration, and exploration was negative (although a stone could of course have been overlooked), the common duct was not opened. A No. 20 F. soft rubber catheter was sewn into the open cystic duct for drainage.

During the course of the dissection of the gallbladder from its bed, an orifice, about two millimeters in diameter, was noticed in the upper portion of the bare area. It was surrounded by a thin but well defined greyish wall. From the mouth of this duct in the gallbladder bed there escaped freely dark, viscid bile, identical with that in the

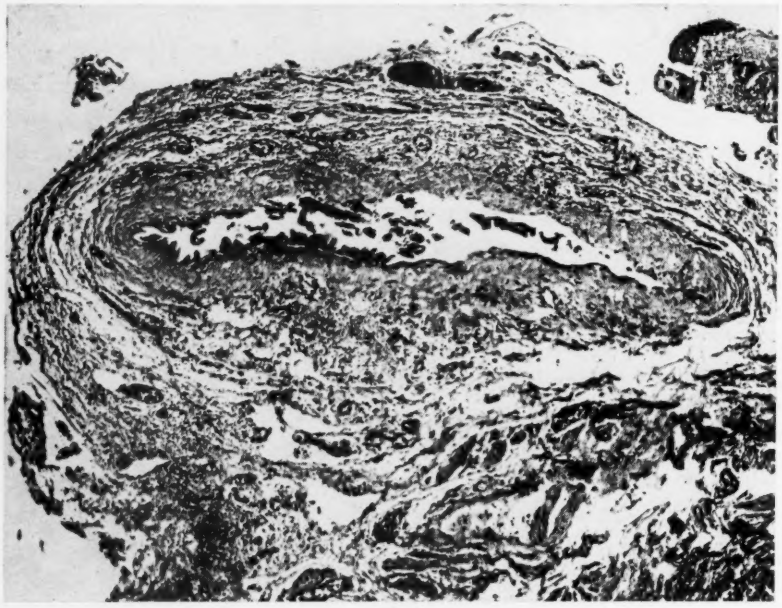

FIG. 1.—Path. No. P14919: Section of cholecystohepatic duct described in Case 1. Structural details are typical of small bile duct.

gallbladder and cystic duct. Following the escape of this dark fluid, thin yellow bile appeared. A fine probe was passed into the orifice and entered a duct in the liver substance for a distance of two to three centimeters in the direction of the porta hepatis. Sections of the duct were removed for microscopic examination. No other anomaly of the extrahepatic biliary system was noted.

The orifice of the anomalous duct was left open. Rubber dam drains were placed in Morison's pouch and also to the mouth of the aberrant duct, in order to insure external drainage of any biliary leakage that might occur. The wound was then closed in layers.

Microscopic examination revealed the specimen to be a typical bile duct (Fig. 1).

Postoperative Course: The immediate postoperative course was uneventful. By the fifth day fever had subsided, icterus had disappeared, the urine was free from bile, and stools were of normal color. The tube in the cystic duct drained thin yellow bile for one week. It was then spontaneously extruded, and some bile discharged into the dressings. The rubber dams were withdrawn several days later.

Eleven days after operation the temperature rose to 102° F. In the absence of any other apparent cause for pyrexia, the fever was assumed to be due to retained infrahepatic biliary seepage. Accordingly, a tube was placed into the small sinus that

remained, and bile discharged freely. The temperature returned to normal in 36 hours. *It was not until the forty-fifth day after operation that the biliary drainage,* which often was profuse, ceased, and was followed by permanent closure of the wound.

For three weeks after operation the patient complained of anorexia, lassitude, and extreme general asthenia. On several occasions there were brief episodes of marked weakness and vertigo, during which the patient became pale, cold, and clammy, the pulse rapid and thready, and the blood pressure fell to 80/40. There was no associated precordial distress, and no other abnormal physical findings to lead to a diagnosis of pulmonary embolization. The generally debilitated state which existed at the end of three weeks was thought to be due to bile deprivation. The acute episodes were referable either to exaggerations of this state or to pulmonary embolization. Treatment was instituted three weeks after operation. Calcium lactate, yeast, halivar oil, and bile salts were administered, according to the recommendation of Doubilet.[11] Improvement soon followed despite the continuation of biliary discharge; appetite and strength returned; there were no further acute episodes, and the patient was convalescent by the end of the fifth week.

The patient was discharged from the hospital in good condition, seven weeks after operation, with a healed wound. *Follow-up:* There has been no recurrence of symptoms referable to the biliary tract.

COMMENT: The precise nature of the anomalous duct discovered at operation can be inferred from the location of its orifice in the liver bed, and the direction of its course. Because of the features which were noted, the structure can be properly termed a cholecystohepatic duct. Its integral relationship to the biliary system is established not only by the flow of bile from its orifice but also by its histologic appearance.

The abnormal appearance of the bile first noticed in the duct, similar to the pathologic fluid in the gallbladder and common duct, is an interesting feature. It suggests a concurrent stasis and infection of the tributary portion of the liver, conceivably of ascending origin from the gallbladder.

In the absence of common duct obstruction, and in accordance with usual experience, drainage of bile from the cystic duct should have ceased within a week or two. The prolonged biliary discharge may, therefore, reasonably be ascribed to leakage from the open cholecystohepatic duct.

The pronounced asthenia after operation, and perhaps the recurrent episodes of mild shock, can be attributed to prolonged continuous drainage of bile, with consequent loss of electrolytes and faulty vitamin absorption. This etiologic relationship is suggested by the prompt response to a regimen of high salt and vitamin content. Biliary deprivation due to leakage from a severed anomalous duct may, therefore, be of serious import *per se,* aside from possible intra-abdominal complications that might ensue.

Ligation of the duct was discussed at the time of operation. The decision to leave the duct open was made because its caliber was relatively large, the amount of tributary hepatic tissue was in doubt, and the functional state of the liver in this icteric patient was unknown. In retrospect, it can be assumed that ligation would have been safe, and that the postoperative complications would not have ensued.

Case 2.—(C. F. 462649): A 63-year-old female entered the Mt. Sinai Hospital with a history of recurrent right upper quadrant abdominal pain for 40 years. These

attacks had become more frequent during the past two months, and, two weeks before admission, for the first time, were associated with chills and fever for several days. At no time had jaundice been noted. The patient's past history was otherwise not contributory.

Physical Examination: The patient was obese. There was no evidence of icterus. Admission temperature was 100.6° F.; blood pressure 130/86. The abdomen was obese and pendulous. Tenderness and spasticity were present over the right upper quadrant, and an enlarged gallbladder was palpable, moving with respiration. Examination disclosed

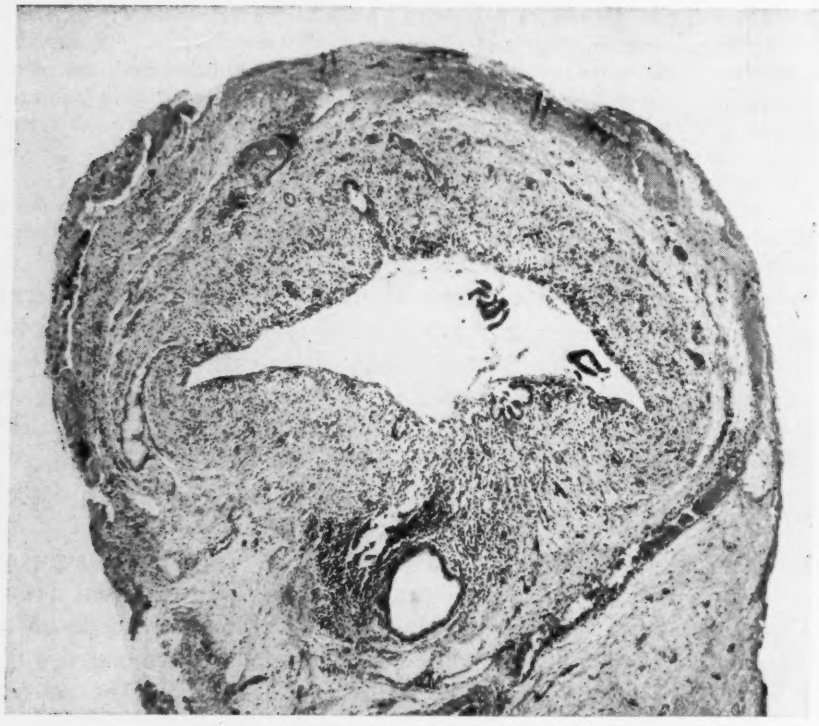

FIG. 2.—Path No. P19088: Section of cholecystohepatic duct described in Case 2.

no other abnormalities. *Preoperative Diagnosis:* Hydrops of the gallbladder. After suitable preparation operation was undertaken.

Operation: A transverse right upper quadrant incision disclosed an enlarged, chronically inflamed gallbladder containing numerous stones. There were many dense adhesions including the cystic and common ducts. A subserosal dissection of the gallbladder was carried out. In the course of the separation of the gallbladder from the liver bed, a sudden escape of bile was noted. Examination of the under surface of the liver, at this point, showed the biliary leakage to occur from the lumen of a duct apparently running into the gallbladder from the liver. A section of the walls of this orifice was taken for microscopic study, and the lumen was then closed with a suture.

Microscopic examination of the aberrant duct section revealed a typical bile duct (Fig. 2).

Postoperative Course: The postoperative course was uneventful. Sutures and drains were removed on the eighth postoperative day. The patient was discharged from the hospital four days later, afebrile, with wound healing well. *Follow-up:* There have been no symptoms referable to the biliary tract.

COMMENT: From the gross appearance of the aberrant duct leaking bile in the liver bed, and its microscopic appearance, there can be no doubt of its nature. The similarity in size and location of this duct with that described in Case 1 suggested the probability of a complicated convalescence, unless ligation was performed. In the absence of evidenct icterus, to suggest hepatic dysfunction, no hesitancy was felt in doing so. Closure was, therefore, done—and an uneventful postoperative course ensued.

CONCLUSIONS

The existence and surgical significance of an anomalous duct between liver and gallbladder (to which we have attached the term "cholecystohepatic" duct) can be understood best by a consideration of its embryology.

Although a duct of substantial proportions probably is a rare anomaly, its presence at the operation of cholecystectomy creates an important problem.

Since the duct is inevitably severed during cholecystectomy, its nonrecognition, because of uncontrolled leakage of bile, may lead to peritonitis, localized infection, or the symptoms of prolonged biliary deprivation.

Ligation of the duct is probably a safe procedure in most cases. In the presence of stasis and infection within the segment of liver drained by the duct, drainage and not ligation may be indicated despite the complications which are invited.

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COMPLICATIONS OF INTRA-OSSEOUS THERAPY

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IN 1940, WHEN the technic for infusing blood or other fluids into the circulation *via* the bone marrow was discussed, the following points were stressed: (a) The method is indicated only when intravenous injections or infusions are needed and the peripheral veins are not available for one reason or another (poor development; delirious or uncoöperative patients; extensive burns; shock). (b) The operator should familiarize himself with the anatomic landmarks in adults (sternum) and infants (tibia and femur), and practice the technical steps on the cadaver before attempting to carry out the procedure in a patient. (c) No irritating substances should be introduced by this route. (d) In the presence of extensive infection, with or without bacteriemia, the use of this route is not advisable except for the introduction of bacteriostatic drugs (sulfonamides, penicillin).[1, 2, 3]

There has been wide application of this method and the reports thus far published have, on the whole, been favorable.[4–19] The feeling may grow, however, that infusion by this method may be undertaken by any one, without previous training and in disregard of the points enumerated above. Lest this happen, attention is hereby drawn to certain serious complications which have resulted from trials at the performance of this technic.

The outstanding example of the consequences of flagrant disregard of simple precautions is that reported by Ravitch,[20] in September, 1943: Seventy-five cubic centimeters of *seven-day-old blood,* removed from a flask *opened two days previously,* were given in the *sternum of an eight-month-old infant.* The child developed a mediastinal abscess requiring drainage, which was followed by recovery. In October, 1941, the anatomic features of the bone marrow in the sternum, femur and tibia of infants were reviewed, and attention was drawn to the fact that in infants under three years of age, the sternum should not be used for this purpose because of its small size and somewhat irregular distribution of its marrow deposits.[3] Use of either femur or tibia was recommended in such patients.

Papper[7] has recorded a death presumably resulting from the administration of 5 per cent glucose solution *via* the marrow of the corpus sterni in a 20-year-old woman, with acute thrombopenic purpura. The infusion needle was inserted into the sternum while a splenectomy was in progress and, because of the necessity of not interfering with the operative field, the needle was pointed caudad. A small amount of marrow was obtained before the

infusion was carried out. The patient died 12 hours postoperatively, and postmortem aspiration of the pleural spaces yielded about two liters of fluid containing glucose in almost 5 per cent concentration. Autopsy was not completed. Whether the insertion of the needle in the caudad direction affected the technic is uncertain.

Elsewhere[21] we have described a similar complication which, however, did not end fatally. In attempting to enter the sternal marrow, two orifices had been made. At the first trial the posterior plate of the sternum had apparently been penetrated. The needle had been withdrawn and the second insertion made, slightly below the first one, but in the same segment of bone. Some of the fluid infused through the point of the second insertion, apparently found its way into the chest through the orifice made on the posterior plate during the first trial (Fig. 6).

Two instances of supposed osteomyelitis have been called to our attention, following attempts at infusion *via* the tibial marrow. In both instances, in spite of the fact that no marrow was removed, an attempt had been made to inject blood forcibly. Each patient received between two and five cubic centimeters of blood. Roentgenograms disclosed elevation of the periosteum and some resorption of the nearby bone in each patient. It is likely that in each of these instances the blood was injected not into the marrow cavity but subperiosteally. The bone resorption changes usually demonstrated roentgenologically[4] in these and similar instances probably result from pressure necrosis rather than from true osteomyelitis. A severe osteomyelitis did follow the use of this route in an infant with *Staphylococcus aureus* abscesses throughout the body, and a probable bacteriemia (ref. 22, footnote). Blood had been injected through the tibia during the height of the acute septic process, not far from an area where there were several subcutaneous abscesses.

Behr[23] gave 60 infusions to infants, and had two complications. In one child the needle-guard pressed against the leg and caused necrosis of the skin. In another child the needle was left in place for about three days, resulting in leakage of fluid around the needle into the subcutaneous space. An osteomyelitis developed which cleared up after surgical treatment. Maintenance of a needle in the bone of infants for prolonged periods of time (over 12 hours) seems inadvisable. It is perhaps preferable to use another bone if the infusion must be repeated every other day. The devices introduced by Gimson,[11] and Behr,[23] to prevent undue motion of the infant's leg while the infusion is in progress and to guard against a too deep penetration of the needle into the bone, are useful additions to the technic.

Meola[12] describes the occurrence of black discoloration of the foot and leg of a six-week-old female child, within one-half hour after 50 cc. of plasma and 50 cc. of 10 per cent glucose solution had been given without difficulty into the right tibia. The discoloration extended one-third of the way up the leg to a point two inches distal to the site of the needle puncture, and was followed by loss of the great and middle toes on the involved foot. The cause

of the reaction was not clear; it was felt that arterial thrombosis might have occurred. Dickins and Richmond[24] have recently reported gangrene of toes associated with a "thrombophlebitis" in a premature infant, and possibly related to the hypodermic injection of a vitamin K preparation into the thigh. This incident, in which the injection was not intra-osseous, and not so intended, necessitated disarticulation of all the metatarsal bones in the involved foot.

The following complication arose after infusions into the sternum of an adult: A 25-year-old woman was hospitalized because of intestinal obstruction. She had had diabetes mellitus for 14 years, and had had her right kidney

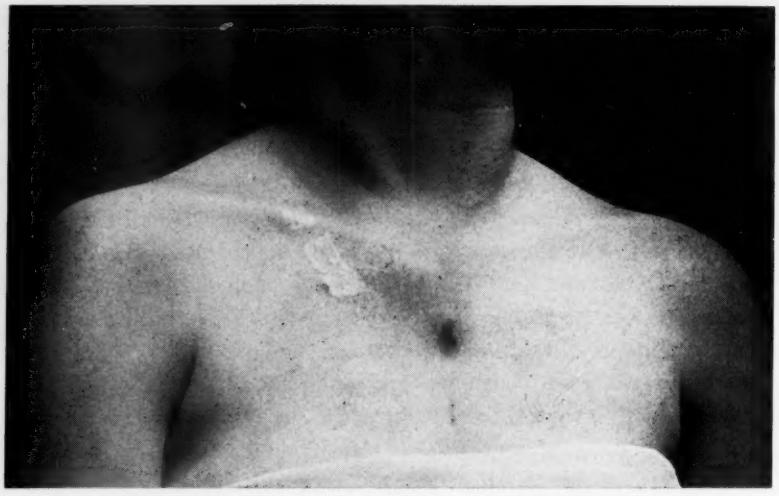


FIG. 1.—Appearance of lesion before incision and drainage.

removed in April, 1943, because of abscesses and pyelonephritis. She later became pregnant and, because of poor renal function and almost unmanageable diabetes, a therapeutic abortion and fundectomy were performed in March, 1944. In June, 1944, she suffered an attack of amebic colitis which responded promptly to therapy.

The intestinal obstruction failed to be relieved by conservative therapy and celiotomy was performed on August 4, 1944, revealing organic, sigmoidal obstruction due to old diffuse pelvic inflammation, with matting together of intestinal loops. The involved loops were freed, cecostomy was performed, and the abdomen closed. Postoperatively, it was difficult to control her fluid balance and diabetes. Feeding during the first few days had to be entirely parenteral. Her veins were extremely small and efforts were made to preserve the few which were available as sources of blood samples needed for biochemical determinations. She received infusions of blood, plasma, 0.85 per cent NaCl, and 10 per cent glucose in saline by the intravenous and intramedullary routes. On August 4 and 5, 1944, the patient received 1,500 cc. of 10 per cent dextrose, 500 cc. of whole blood and 500 cc. of 0.85 per cent NaCl *via* the

marrow of the corpus sterni. On August 12, 1944, while receiving whole blood *via* the marrow of the manubrium, the needle worked loose and before its malposition was discovered, a fairly large amount of blood had escaped into the subcutaneous tissues of the upper anterior chest wall. Her postoperative course was further complicated by wound disruption and a fecal fistula. The wound was resutured and the fistula gradually closed. Because

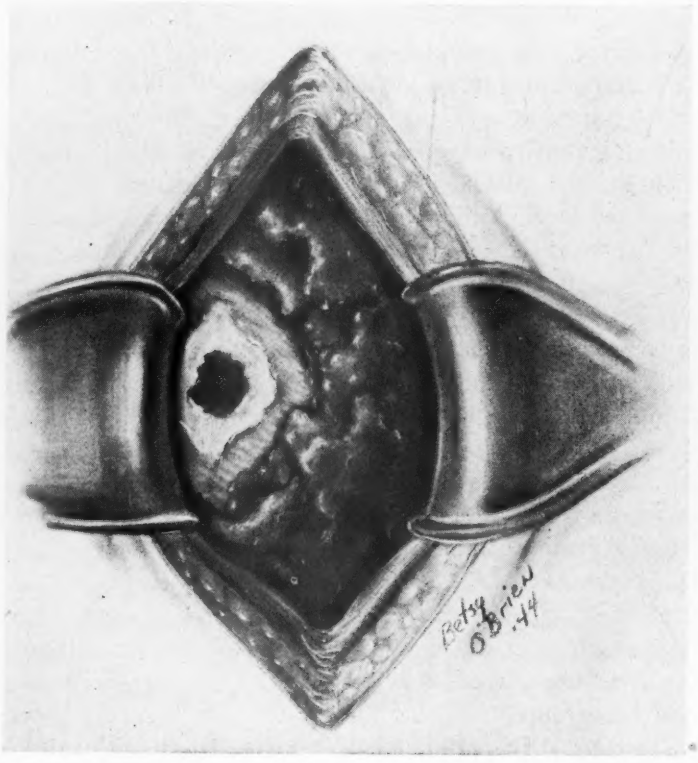


FIG. 2.—Operative exposure of the site of the infusion into the sternum.

of the deposition of blood in the subcutaneous tissues, the area over the manubrium sterni was diffusely swollen and discolored. This reaction gradually decreased, leaving a 4-cm. zone of induration, which was elevated 0.5 cm. above the surrounding skin. This area remained slightly tender, and at irregular intervals the patient complained of pain in her right shoulder top. Clinical and roentgenologic search failed to disclose any intrathoracic or subphrenic lesion.

The patient was discharged, September 30, 1944, and five days later the area over the manubrium gradually began to increase in size and in tenderness. At the same time the dull pain in her right shoulder grew worse. The swollen zone pointed and opened spontaneously, draining thick yellow pus. She was treated by her local physician and returned to the clinic for reexamination one month later. At that time the two sinuses were draining purulent

material and the surrounding zone was slightly swollen and tender (Fig. 1). Culture of the exudate yielded *Staphylococcus albus*. Roentgenologic examination of the sternum disclosed slight irregularity about the right lateral margin of the manubrium near the sternoclavicular articulation, with localized areas of decalcification and irregularity of the cortex, due probably to osteomyelitis.

On November 9, 1944, under cyclopropane-oxygen anesthesia, a vertical incision was made through the sinus tracts and carried down to the manubrium sterni. A core of soft granulation tissue extended from the skin sinuses down to a 4-mm. opening in the anterior sternal plate (Fig. 2). The opening was enlarged, exposing an area of seminecrotic tissue, which was curetted away. The entire cavity was packed with iodoform gauze. Tissue removed at operation showed inflammatory and fibrotic reactions, with a few bony spicules scattered through the sections. When discharged from the hospital, November 15, 1944, the cavity was clean and draining little. She is being cared for by her family doctor and his last communication states that the wound was healing well.

The advanced deterioration of the patient's metabolic status probably contributed somewhat to the local changes at the point of the infusion. The same reason, however, made it imperative, originally, to use the intra-osseous route, since peripheral veins were unavailable. In this group of patients one must, therefore, take special precautions to avoid mishaps such as dislodgement of the needle, long maintenance of the needle in the bone, and possibility of surface contamination. A similar complication has been recorded by Jimenez Pinto.[4]

COMMENT: A distorted impression of the risks of intra-osseous therapy is certain to result from isolated consideration of the above-mentioned complications. A more balanced view is obtained if the complications are viewed against the background of those known to have followed other types of parenteral therapy. The intramedullary route shares with other parenteral routes the fundamental risks and complications surrounding the introduction of a hollow needle into the body. In addition, it has a few risks of its own. In many respects, the present status of intramedullary injections is analogous to that of intravenous, intramuscular and subcutaneous injections during the second and third decades of the present century. The medical literature of that period contains numerous references to the complications and difficulties experienced at a time when certain parenteral routes were becoming popular, but were still under trial. No figures are available for the average incidence of complications which occurred either during or after such injection therapy and whether they resulted from action of the substance injected, faulty technic, or both. Local suppuration as well as aseptic necrosis following parenteral injection (intravenous, intramuscular or subcutaneous) was reported by various authors.[25-28] Gas gangrene following injections was discussed by Junghanns,[29] and Harney.[30] The latter author reported 86 instances of gas gangrene following various parenteral injections; the mortality

for this group was 88 per cent. Reports of venous thrombosis and thrombophlebitis following injection therapy, leading at times to fatal pulmonary emboli and pulmonary infarctions, were not uncommon.[31–36] Systemic infection, including septicemia and metastatic osteomyelitis, was reported by Gants.[32] Various types of nerve injury followed injection therapy.[37–40] Payenneville and Castagnol[41] recorded an instance of intramuscular injection in the gluteal region producing gangrenous changes and destruction of the

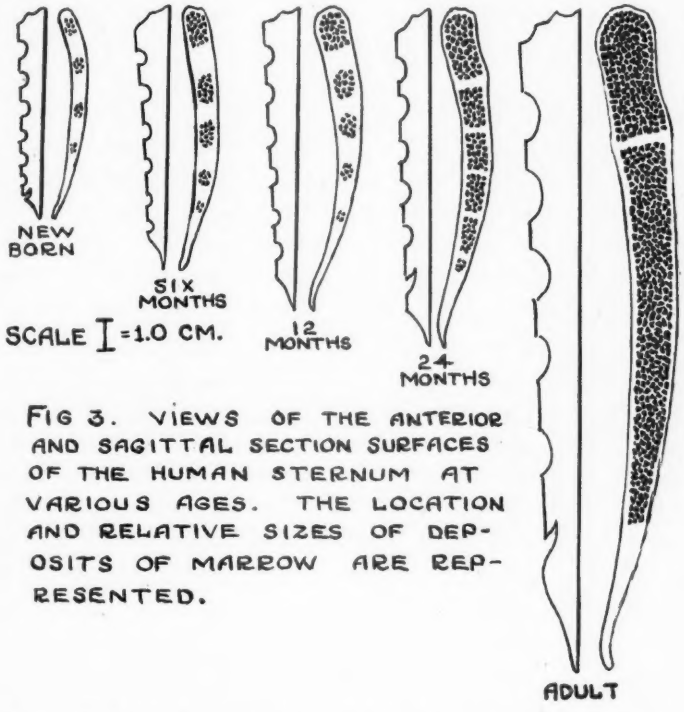


FIG. 3.—Diagrammatic representation of the relative sizes of human sternal plates, showing the development of marrow deposits.

anastomosis of the gluteal and internal pudendal arteries, with necrosis and gangrene of the rectum, bladder, genitalia and thigh. Arterial embolism has followed the intramuscular injection of iodobismotol into the buttock.[42] Complications, both local and general, have followed intra-arterial injections for roentgenographic visualization of the vessels.[43]

Therefore, when assessing the dangers of intra-osseous therapy, one must keep in mind the risks inherent in the parenteral administration of any substance, in addition to those that may be peculiar to intra-osseous therapy. Our own experience with over 400 intra-osseous infusions, and the experience of others, [1–23] have not caused us to modify our feeling that, if the method is employed only when indicated, by those who are familiar with the technic, and who observe the known contraindications, the risks involved do not appear to be any greater than those carried by other forms of parenteral therapy.

SUGGESTIONS FOR THE PREVENTION OF COMPLICATIONS

Aside from complications caused by bacterial contamination, most difficulties seem to originate from failure to consider anatomic details, and how they govern the fate of fluid injected into various bone marrow deposits.

The Sternum in Infants.—At birth, the sternum is a cartilaginous plate 5 to 7 cm. long, from 1.0 to 1.5 cm. broad, and from 1.0 to 3.0 mm. thick. There are usually three deposits of marrow in its substance, varying in diameter from 5.0 mm. in the manubrium (which is the most constant one in size and location) down to 1 or 2 mm. in the lower part of the body (Fig. 3).

As the infant grows, these deposits of marrow increase in size. By the age of 24 months the original island of marrow in the manubrium occupies all of that part of the sternum save for a soft, partly ossified, but mainly cartilaginous cortex, about 0.5 mm. in thickness. It is important to note, that, in the specimens so far examined, in no instance did the marrow in the manubrium communicate with that in the corpus sterni (Fig. 3). Any attempt to infuse fluid into the sternal marrow of an infant less than 24 months old is courting almost certain failure or disaster, for: (a) the marrow islands are small; (b) they are irregular in location; (c) the sternal plate is soft and only 2 or 3 mm. thick, making entrance into the mediastinum highly probable; and (d) even if the needle tip fortuitously rested in a marrow deposit, its small size would prevent its transporting any appreciable amount of fluid. Theoretically, at the age of 24 months, one might infuse fluid *via* the manubrial marrow without complication if the operator were unusually dexterous, but, because of the above-mentioned reasons, injections in any part of the sternum in a child less than 36 months old should never be attempted.

The Tibia and Femur of Infants.—The lower end of the femur and the upper end of the tibia are the sites of election for the infusion of fluid in infants up to four, and probably up to five years of age. The marrow deposit in the distal end of the femur is approximately 1.8 cm. broad at its widest point, and 6 to 10 mm. deep, while that in the proximal tibia is usually 6 to 8 mm. in any diameter. At birth, the distal 1.0 to 1.5 cm. of the femur and the proximal 1.0 to 1.5 cm. of the tibia are cartilaginous, and each contains a single center of ossification. These centers appear at about the time of birth, and join their respective shafts at about the twentieth year.

In infancy the patella is cartilaginous and ossifies from a single center, which appears at the third year and is completely ossified at puberty. When inserting a needle into the marrow cavity of the lower end of the femur in an infant, the operator must avoid the patella, and he must recall that the needle will traverse the upper extension of the synovial cavity beneath the quadriceps tendon anterior to the femur (Fig. 4). Careless technic may result in injury to the epiphyseal line or causing fluid to enter the knee joint.

The marrow cavity of the upper end of the tibia must be entered, not

directly anteriorly, but on the anteromedial surface of the bone, with the needle pointed away from the knee joint, in order to avoid injury to the epiphyseal line, which lies 1 to 1.5 cm. distal to the distal edge of the patella when the knee joint is extended (Fig. 4).

When inserting a needle into either tibia or femur, it is possible to drive the point through both anterior and posterior cortical plates, with resulting deposition of fluid into the popliteal space. This area is enclosed by fascia

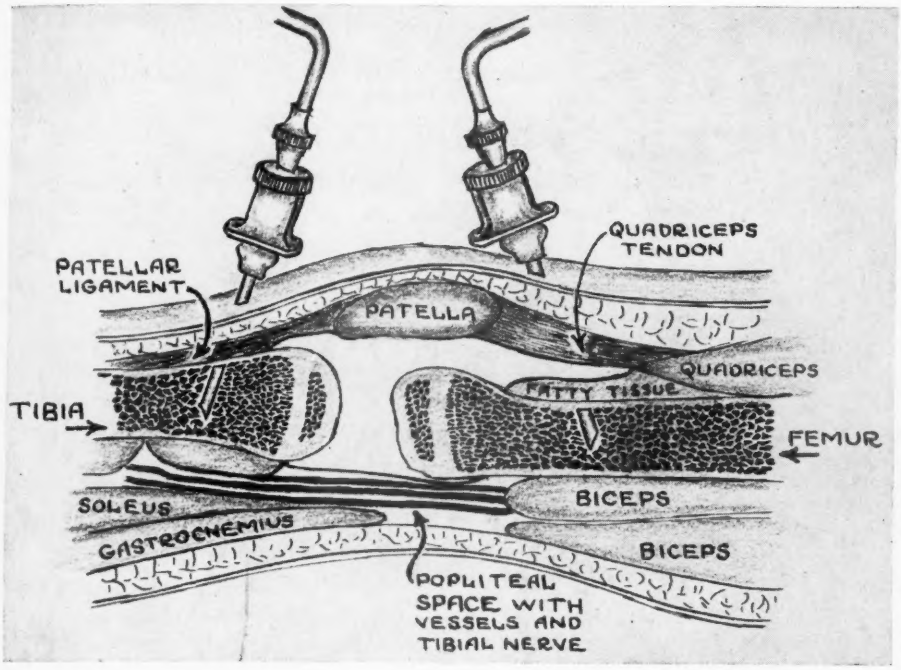


FIG. 4.—Diagram of a longitudinal section of the knee joint area in a full-term, newborn infant, showing the important anatomic relations and the correct position of needles for infusion. In the diagram the size of the joint cavity proper is purposely exaggerated.

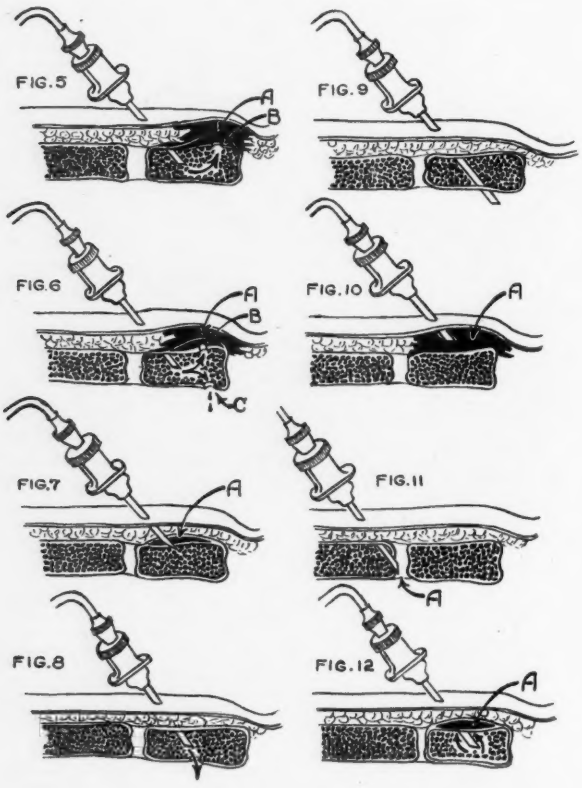
and sufficient pressure may be built up within it (particularly if the substance is injected rather than administered by gravity flow) that the popliteal vessels may be shut off and the tibial nerve damaged. The needle-guard and precautions advocated by Gimson[11] should help in preventing this mishap.

The red marrow in the tibia and femur begins to change into fatty marrow between the fifth and seventh year. Theoretically, therefore, the tibia or femur should not be used for infusion after the age of five.

The Adult Sternum.—If a needle is inserted into either the manubrium or the corpus sterni and, for any reason, the infusion attempt is unsuccessful, under no circumstances should the needle be removed and reinserted close by the first puncture site within 12 hours. This is most important when any suspicion exists that the posterior plate might have been punctured. If this precaution is overlooked and a second puncture is made soon after, near to the preceding orifice, part of the fluid infused *via* the second orifice will

leak out through the first one, resulting in a collection of fluid either beneath the skin, beneath the periosteum (Figs. 5 and 6-A, B) or in the mediastinum (Fig. 6-C).

A similar word of caution applies to infusing fluid through a needle which has been deeply inserted, and then partially withdrawn after the operator was unable to aspirate marrow. This maneuver may leave a hole in the posterior surface of the sternum, and fluid infused will leak into the



FIGS. 5 to 12, incl.—Mechanism of complications resulting from various errors in the technic of inserting a needle into the sternal marrow. Each drawing represents a sagittal section of the manubrium and upper portion of the body of the sternum of adult man.

mediastinal space (Fig. 8). If the needle tip perforates the posterior sternal plate the operator will not be able to aspirate marrow (Fig. 9).

When, for any reason, a needle is withdrawn from one compartment of the sternum, and must be reinserted soon thereafter, it should be placed in a separate portion of the sternum. Thus, if the first attempt was in the manubrium, the second should be made in the corpus, or *vice versa*. It is probably safe, when the need is imperative, to make more than one puncture in the body of the sternum within a short time (two to three hours), provided the orifices are at least six or seven centimeters apart.

If sufficient time has elapsed for previously made orifices to become occluded by firm clots, a second puncture can apparently be made nearby with safety. It is assumed that 12 hours is sufficient time for a firm clot to form unless a disorder of the blood is present. This time-interval will also vary with the amount of pressure applied to the fluid infused through the second orifice.

If a long-bevelled needle is used, it is possible for some of the infused fluid to enter the marrow cavity and some to leak out subperiosteally (Fig. 7). A similar difficulty arises if the needle works loose, and fluid leaks out around

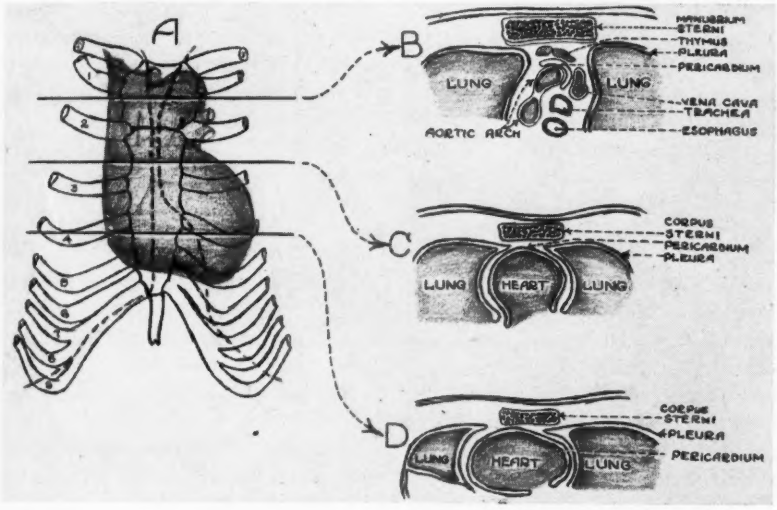


FIG. 13.—Diagrammatic representation of cross-sections of the sternum and thoracic viscera made at the three levels at which intra-osseous infusions are usually given. In "A" the solid dark shadow represents the outline of the heart and great vessels; the solid black lines represent the outlines of the sternum and adjacent parts of the clavicles and costal cartilages; the dotted lines represent the reflections of the pleura. From the cross-section diagrams, "B", "C", and "D", it may be seen how faulty technic might result in damage to the intrathoracic viscera.

it (Fig. 12). A needle may become dislodged completely from the bone, and a quantity of fluid may be deposited in the subcutaneous tissues (Fig. 10). This occurred in the patient whose case was related above. If a needle is inserted too high in the corpus sterni, or at too oblique an angle, its point may come to rest in the cartilage of the superior intersternal articulation. In such an instance, no marrow would be aspirated, and, therefore, an infusion should not be attempted (Fig. 11).

From a consideration of the cross-sectional anatomy of the sternum and thoracic viscera, as diagrammed in Figure 13, it is obvious that faulty technic may result in instillation of fluid into the pleural, pericardial or mediastinal spaces, with sequelae depending on the amount and type of fluid infused.

SUMMARY

To avoid needless complications, the intra-osseous route for the infusion of fluids should be employed only when indicated, by those acquainted with

the technic and the existing contraindications. The risks involved in intraosseous therapy do not appear to be any greater than those inherent in other forms of parenteral therapy. A complication is reported following infusion *via* the sternal marrow of a diabetic. The relevant anatomy, as well as certain actual and possible technical mishaps, are reviewed.

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COMA DURING AND FOLLOWING SPINAL ANESTHESIA

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IT HAS LONG BEEN KNOWN that severe anoxemia produces damage to the brain tissues, the cortical areas being the most vulnerable. Caine[1] first suggested the possibility of cerebral damage as the cause of death following nitrous oxide-oxygen anesthesia. Courville[2], in his classical report, showed that the pathologic changes in the brain in deaths following nitrous oxide anesthesia were similar to those found after asphyxia, and, thus, definitely linked the etiology to anoxemia.

Many other investigators have shown this to be true in methods of anesthesia other than that with nitrous oxide. Steegman[3] reported four cases of prolonged anoxemia, two following nitrous oxide, one after avertin, and one with cyclopropane. The clinical syndrome in all cases was characterized by coma or stupor interrupted by screaming, maniacal delirium, hyperkinetic restless motor symptoms, visual disturbances and autonomic signs: variability in the pulse and blood pressure. Murphy[4] reported a case of severe nueropsychiatric disability following partial exsanguination and shock from blood loss following an attempted suicide. There was no anesthetic involved in this case. Schnedorf[5] *et al.*, report two cases, one, a man who went into shock during an extensive operative procedure under spinal anesthesia, lapsed into coma, and remained there until death. The other was a child who received open-drop ether anesthesia for approximately one hour and then did not react, but instead showed signs of brain damage and died. Both these cases, on autopsy, showed the cortical degeneration typical of anoxemia.

It has been demonstrated experimentally and seen clinically that the extent of brain damage is directly proportional to the duration and severity of the anoxemia[6, 7, 8]. In this report, anoxia of sufficient extent to produce coma during spinal anesthesia will be considered. In all these cases collapse and coma occurred, but prompt intervention made possible complete recovery of the patient. The relief of the anoxia prevented the occurrence of irreversible changes in the brain.

ETIOLOGY

The etiology of anoxia in spinal anesthesia is varied. Three chief factors are involved. If the anesthesia reaches too high a level, the resultant paralysis of the intercostal muscles causes a decrease in the vital capacity. The impaired respiratory excursions lead to decreased oxygenation, which, if not counteracted with inhalations of high concentrations of oxyen, leads to fatal anoxia. If the level of anesthesia reaches inordinate heights, it may involve the diaphragm, in which case respirations must be maintained by artificial means such as intermittent manual compression of the rebreathing bag of an anesthesia machine or artificial respiration by any of the accepted methods

of manual compression of the chest, such as the Schaeffer. The former is the better since efficient ventilation with high oxygen concentrations can be attained.

Secondly, the drop in blood pressure associated with any of the factors in an operative procedure, such as hemorrhage, reflexes from traction on the mesentery, or the vasodilatation of the spinal anesthesia itself, if allowed to persist, leads to surgical shock. The hypotension results in severe anoxia of the cerebral centers. When the respiratory center is subjected to acute anoxemia it may be markedly depressed, resulting in apnea of central origin.

Third, mechanical factors may cause anoxia from impaired respiratory excursions. The use of abdominal packs against the diaphragm, retractors against the costal margins, assistants leaning on the chest, steep Trendelenburg position, and kidney or gallbladder bars, all tend to inhibit respiratory excursions and may lead to anoxic anoxia intense enough to precipitate a collapse.

Anoxia, when severe, produces unconsciousness for varying periods of time, and the effects may be divided into four groups, depending upon the severity of the anoxia and the changes it produces. When remedied it may resemble syncope, with rapid recovery of consciousness. In cases where the anoxia has persisted for a slightly longer time, the patient may remain unconscious for periods up to 24 or 36 hours, but recover without sequelae. After sublethal periods of anoxia, the patient eventually recovers consciousness and lives, but shows signs of permanent brain damage. In the final group, the patient either dies on the operating table or remains comatose in the postoperative period, with signs of cortical degeneration until death occurs in several days.

The following case reports illustrate several of the etiologic factors and the treatment of the anoxia when it occurs.

CASE REPORTS

Case 1.—J. B., a well-developed, moderately obese, 50-year-old, white female, was admitted with acute appendicitis. Premedication of morphine sulphate gr. ¼, and atropine sulphate gr. 1/150 was given subcutaneously 15 minutes before the anesthetic. Ephedrine sulphate, gr. ¾ was given hypodermically five minutes before the spinal tap.

Lumbar puncture was performed in the third interspace, with the patient in the left lateral position. Two cubic centimeters of spinal fluid were withdrawn and used to dissolve 100 mg. of procaine hydrochloride, to which was then added 1 cc. (10 mg.) of pontocaine, and the mixture injected slowly. The patient was turned upon her back in 5° Trendelenburg, and the operative procedure was started five minutes later. The blood pressure was still at the preoperative level of 130/80. When the appendix was being removed ten minutes later, the patient complained that she could not catch her breath and then rapidly became cyanotic. At this time her pulse was rapid, and the blood pressure 170/110. The patient ceased voluntary respirations several minutes later, and 100% oxygen was administered by manual compression of the rebreathing bag of the anesthesia machine. The patient was now unconscious and did not respond to any stimuli. Her blood pressure rose to 220/120, and the operation was rapidly concluded. Intravenous medication of coramine, Gm. 0.25 and caffeine sodium benzoate gr. 5 were given while the patient was being maintained with artificial respiration. Spontaneous

respirations were resumed after about five minutes of artificial respiration but were diaphragmatic and very shallow and were reinforced by manual pressure on the rebreathing bag for about 30 minutes.

In the hour immediately following the operation, the blood pressure gradually fell from its high of 220/120 to its approximate normal of 120/70, and remained there. The patient was still comatose, not responding to any stimulation. Bilateral positive Babinski signs were present.

After two hours the oxygen was discontinued since the patient's color was good and the pulse and blood pressure well within normal limits. Since the patient was still comatose, a mixture of 95% oxygen and 5% carbon dioxide, was given every hour for ten minutes for the first 20 hours in the postoperative period, and her position in bed changed every two hours. The first response to stimuli was obtained 16 hours after the patient had lapsed into coma, and she finally spoke coherently, knew who and where she was, 30 hours after operation. She then had an uneventful and uncomplicated recovery. No sequelae remained.

COMMENT: The cause of the collapse in this case apparently was a spread of the anesthesia to too high a level, first involving the intercostal muscles and then reducing the vital capacity, and then involving the phrenic nerve causing paralysis of the diaphragm. The resultant apnea produced anoxic anoxia which was combated by ventilation with 100 per cent oxygen by artificial respiration. The pulse and blood pressure remained strong and responded to the anoxia by trying to supply more blood to the brain as evidenced by the increased pulse rate and rise in blood pressure. If the anoxia had not been relieved, the blood pressure would have subsequently fallen before the death of the patient.

Case 2.—T. D., a thin, 52-year-old, white female, in poor general condition, was admitted with an abdominal abscess, which was drained under general anesthesia, with cyclopropane, oxygen and ether. Two weeks later she showed signs of intestinal obstruction, and a barium enema revealed a growth in the rectosigmoid region. A cecostomy was performed under spinal anesthesia, using 100 mg. of monocaine formate in 2 cc. of spinal fluid injected into the third lumbar interspace. The patient tolerated the procedure well, with no change in condition.

After she was given strong supportive treatment and her general condition improved, she was scheduled for resection of the rectosigmoid. Premedication was seconal gr. 3 *per os* at 6:30 A.M. and morphine sulphate gr. 1/6 and atropine sulphate gr. 1/150 subcutaneously at 7:30 A.M. Continuous spinal anesthesia was started at 8:25 A.M., and the incision made at 8:35 A.M. She was given 3 mg. of neosynephrin subcutaneously at 8:20, and an infusion started at 8:30. During the operative procedure she received 1,000 cc. of saline and 500 cc. of whole blood. The anesthetic agent was procaine hydrochloride injected into the third lumbar interspace, with fractional doses of 120 mg. first dose and 60 mg. each succeeding dose for a total of 480 mg. The operation proceeded nicely, and the patient's condition remained fairly good, the pulse varying from 72 to 88, and the blood pressure after falling from 120/70 to 80/56 in the first half hour remained there throughout the procedure.

At 12:10 P.M. the blood pressure fell to 65/46, and coramine Gm. 1.25 was given intravenously. At 12:15, while the abdomen was being closed, the patient went into severe shock. Blood pressure, pulse and respirations were absent; the skin was cold and clammy. The patient had been receiving a 50% mixture of nitrous oxide and oxygen throughout the operative procedure, and now this was changed to 100% oxygen and given by artificial respiration, using the rebreathing bag of the anesthesia machine. The

following stimulants were given intravenously into the tubing of the infusion: ephedrine sulphate gr. ⅜, caffeine sodium benzoate gr. 7.5, metrazol gr. 3. At 12:30 P.M. the pulse had returned and was 104. The blood pressure was 100/76. At 12:45 the blood pressure was 140/100, pulse 132. The operation was concluded, and the patient returned to her room.

The patient had been awake and able to respond throughout the operative procedure until the time she went into shock. At this time her respirations ceased along with the disappearance of the pulse and blood pressure. She remained unconscious although the respirations were resumed in eight minutes, and she did not respond when returned to the ward, where she was given 500 cc. of plasma and oxygen by B.L.B. mask. Her breathing was stertorous and general condition poor. At 1 o'clock she had a mild convulsive seizure which started in the face and arms and spread throughout the body. These seizures occurred intermittently for about an hour, each lasting for several minutes. Her eyes were staring, glassy; there was no corneal reflex. Her blood pressure rose during the afternoon to 156/60 at 5 o'clock. She vomited once at about 5:30, but still did not respond. At 6:00 o'clock the blood pressure had risen to 160/86, breathing was easier, and the patient was sleeping fairly quietly. At 6:30 she had another convulsive seizure which lasted about ten minutes. Neurologic examination at this time showed spasticity of the upper extremities and flaccidity of the lower extremities. She started making involuntary movements at 9:00 o'clock in the evening and voided in bed at 11:30. At 12:30 A.M. she responded slightly, fixing her eyes upon the person speaking to her. Blood pressure at this time was 150/70. Throughout the night she gradually recovered some of her responses, awakening at times and responding to stimuli. In the morning she was able to speak a little and her restlessness had decreased. She felt no pain in the operative area and was still getting oxygen *via* the B.L.B. mask. At 9:00 A.M. the oxygen was discontinued since she was feeling much better and responded well to questioning. The blood pressure was now 148/74. During the day it gradually fell to its normal level of 120/70. Subsequently she had an uneventful recovery, and was discharged from the hospital in good condition.

COMMENT: This is an example of cardiovascular collapse following a prolonged major operative procedure where the patient had been carried along on the borderline for several hours. At the close of the operation the patient could no longer tolerate the lowered blood pressure, which itself was being maintained by the body reflexes brought into play to maintain circulating blood volume, and peripheral collapse followed. This might have been prevented by the administration of vasopressor drugs when the last drop in blood pressure to 64/46 was noted. Ephedrine sulphate in place of coramine would probably have produced better results.

The important feature is that once the collapse did occur, vigorous measures were undertaken at once to stimulate the cardiorespiratory system. An improvement was obtained in several minutes, so that the pulse, respirations and blood pressure were within normal limits. The interval, however, had been long enough to produce a moderate amount of reversible brain damage.

The etiology of the interference with respiration in this instance is central. The severe drop in blood pressure caused an ischemia of the respiratory center, producing an apnea of central origin. The mechanical respiratory apparatus was still intact and capable of functioning, but the regulatory mechanism had been removed. When a sufficient supply of blood and oxygen were again made available for the cells of the respiratory center, it resumed its duties. and normal respirations were restored.

The period of anoxia was at the time limit for reversibility of brain tissue damage. It was enough to produce coma and convulsions, but still the patient recovered without sequelae.

Case 3.—J. A., a well-developed, well-nourished, 17-year-old white male, with intestinal obstruction of three days' duration, due to postoperative adhesions, was to have a celiotomy performed. Premedication was morphine sulphate gr. 1/6 and atropine sulphate gr. 1/150 subcutaneously 45 minutes before operation.

Ten minutes before the anesthetic was administered, the patient was given 3 mg. of neosynephrin hypodermically. Spinal anesthesia with 150 mg. of monocaine was given in the third lumbar interspace, with the patient in the left lateral position. He was immediately turned on his back and placed in slight Trendelenburg. The blood pressure was 120/74, and the pulse rate 88. After an infusion of physiologic saline solution was begun in the left arm, the patient was draped, and the operation started. His condition did not change until the abdomen was opened and traction was exerted upon the bowel in an attempt to expose the site of the obstruction. At this time the patient gasped that he could not breathe and immediately stopped spontaneous respirations. Pulse and blood pressure were absent, and the skin was ashen gray in color and covered with a cold sweat. The surgeon was advised of the change in the patient's condition and asked to release the bowel. Artificial respiration with the rebreathing bag of the anesthesia machine was started, and caffeine sodium benzoate gr. 7.5 and ephedrine sulphate gr. 3/8 were given intravenously into the tubing of the infusion.

In about five minutes the patient again started spontaneous respirations, and the pulse became of good quality although fairly rapid (140). The blood pressure was now 100/66, but the patient was still unconscious and did not respond to stimuli. The operation was continued and carried to a successful conclusion, with no further change in the patient's condition. It was not until the skin was being closed 25 minutes later that he opened his eyes and weakly asked if the operation was nearly over.

An uneventful postoperative course followed, and he was discharged in good condition.

COMMENT: This is an example of shock from a combination of factors—sudden release of intra-abdominal pressure and traction upon a mesentery. These led to sudden peripheral vasomotor collapse, the patient exhibiting typical signs of shock. The prompt treatment restored the cardiorespiratory system, and the period of unconsciousness was only one-half hour, resembling syncope in appearance. The apnea was again the result of sudden severe ischemia of the respiratory center.

Case 4.—J. S., a 50-year-old white mechanic, was admitted to the hospital with a chief complaint of diffuse abdominal pain, more severe on the right side, of ten days' duration, and moderate dyspnea. Examination showed pleural effusion on the right side, later confirmed roentgenologically, and marked tenderness in the right flank. A work-up for intra-abdominal pathology, including barium enema, was negative. The day after admission it was noted that the patient had not voided, and nothing was obtained on catheterization. The next day, when cystoscopy was performed under spinal anesthesia, a flow of urine started. Pyelograms showed poor kidney function and bilateral hydronephrosis, and ureteral catheters were inserted to the kidney pelves for drainage. They drained intermittently for six days, when the patient had a severe chill and drainage stopped completely. A right kidney exploration and possible nephrostomy were decided upon.

Spinal anesthesia was selected, since at the first cystoscopy a flow of urine was obtained immediately following the onset of anesthesia, and it was thought that it might again prove of value. Premedication of morphine sulphate gr. 1/6 and atropine sulphate

gr. 1/150 was given subcutaneously at 4:55 P.M. and at 5:40 P.M. neosynephrin, 5 mg. was injected hypodermically. The blood pressure was now 150/86, and the pulse rate 92. At 5:45 the patient was put in the left lateral position, and monocaine formate 150 mg. in 3 cc. of spinal fluid was administered in the second lumbar interspace. The patient was turned on his back for ten minutes and then, at 5:55, was put in the right kidney position, the table broken to an acute angle, and the kidney-bar raised. The operation was started at 6:05, at which time the blood pressure was 132/80, and pulse rate 96, and the patient was apparently in good condition. At 6:20 he complained of difficulty in breathing and was given 100% oxygen *via* the anesthesia machine. At this time his blood pressure was 100/70, and pulse rate 108, and an infusion was started. At 6:25 respirations ceased, the pulse was very weak, and the blood pressure not obtainable. Artificial respiration with oxygen was given by manual compression of the rebreathing bag of the anesthesia machine. The patient was now cyanotic, and there was difficulty in maintaining a patent air-way due to his position on the operating table. The surgeons were asked to hurry, and drains were inserted into the perinephric abscess which had been found, the table straightened, and the wound quickly closed after a biopsy of the kidney. Ephedrine sulphate gr. 3/8 was given intravenously at 6:28. At 6:35 the patient was put on a stretcher on his back, and although no pulse or blood pressure was obtainable, his color was now fair, and heart beats could be heard with a stethoscope. Artificial respiration was continued, and metrazol gr. 4.5 was given intravenously. At 6:45 spontaneous respirations were resumed, and an intratracheal tube was inserted. The pulse was now obtainable, weak but regular. At 6:50 the pulse was fairly strong, the blood pressure 90/60, and the patient was returned to the ward.

On arrival at the ward at 7 o'clock, the patient appeared to be comatose, skin cold and clammy, blood pressure 108/64, and pulse rate 92. At 8 o'clock his general appearance was better, the skin was warm, the pulse 88, and the blood pressure 110/80. After the intratracheal catheter was suctioned and some mucus obtained, the patient's breathing was easy and not labored. At this time he coughed, and the intratracheal tube was removed. He still did not respond, and no corneal reflexes were present. At 9:30 generalized twitching movements were noticed, with the patient thrashing about moaning and his eyes rolling. At 10 o'clock he still did not respond, but slight corneal reflexes were noted. At 12 o'clock good corneal reflexes were present, and the patient had convulsive movements of his arms and legs for about five minutes. The next morning, at 4 A.M., the patient still did not respond to questioning but reacted slightly to stimuli. At 10 A.M. the patient was very drowsy but answered sluggishly when questioned. At 2 P.M. he was fully aware of his surroundings and seemed to have recovered completely. The second postoperative day his condition was good except for the persistent anuria. He then gradually sank into uremic coma, and died on the fifth postoperative day.

Postmortem examination showed right lower lobe pneumonia, multiple focal infarctions and abscesses of both kidneys, and adenocarcinoma of the stomach with metastases to the abdominal lymph nodes. Contributing factors were hypertrophy of the heart with chronic pericarditis and right hydrothorax, bilateral adhesive pleuritis and atelectasis. Permission for examination of the brain was unfortunately not obtained.

COMMENT: The anoxia and collapse were due to a combination of factors. The respiratory system was already impaired by the pneumonia and pleural effusion. Superimposed upon this was the position of the patient on the operating table, the exaggerated lateral flexion further impairing respiratory excursions, and the level of the spinal anesthesia paralyzing the lower intercostal muscles. The resultant decreased oxygenation rapidly led to collapse through anoxia of the vital centers.

The patient was revived through rapid cooperation between surgeon and

anesthetist in remedying the etiologic factors as soon as possible while instituting the proper resuscitative measures. Artificial respiration with 100 per cent oxygen was immediately started after the patency of the airway was assured. The cardiorespiratory system was stimulated by the injection of ephedrine and metrazol, with good response.

The patient was comatose for approximately 20 hours, and during this time had several convulsive seizures typical of cerebral damage. He recovered fully from the anoxia, however, since 48 hours postoperatively no changes indicative of central nervous system damage could be demonstrated. He subsequently sank into uremic coma and died.

Discussion: Coma during spinal anesthesia is due to a breakdown in the body mechanism supplying oxygen to the brain. In most of the cases presented it assumed the form of acute collapse of the patient and was associated with a sudden fall in blood pressure. The need for an intelligent, experienced anesthetist in charge of the patient's vital functions immediately becomes apparent. If the collapse is recognized as such when it occurs, and the proper treatment instituted with no delay there need rarely be lost a patient under spinal anesthesia. It is when incompetent persons are told to "watch the spinal" and strict attention is not paid to the patient's condition at all times that the collapse is allowed to exist for a period of time that renders irreversible damage. When treatment is instituted too late the patient may linger for some hours or days in coma before expiring. If the critical state of the patient is not recognized at all, the discovery is suddenly made that the patient is dead.

In the treatment of this complication, prophylaxis is the first step to be taken and involves the use of vasopressor drugs as ephedrine sulphate or neosynephrin administered before the spinal. These tend to maintain the blood pressure after the vasodilatory effects of the spinal have occurred. If, in spite of this, the blood pressure falls too low, additional measures must be taken. Small doses of the pressor drugs intravenously will raise the pressure temporarily while intravenous fluid therapy is being started. In any prolonged major operation an infusion should be started before the incision is made. This serves two purposes, providing a means of administering blood or plasma as desired and also keeping a vein open for intravenous medication in an emergency. The fluids thus given also help maintain the circulating blood volume and the patient's blood pressure.

The maintenance of an adequate oxygen intake is a second factor of prime importance. With impaired respiratory effort, the inhalation of high oxygen concentrations (50 per cent to 100 per cent) is sufficient to provide adequate oxygenation. When apnea occurs, however, it is essential that a means of administering artificial respiration be at hand, and there is no better method than one of the closed system anesthetic machines equipped with a rebreathing bag. Intermittent manual compression of the bag will insure alternate expansion and deflation of the lungs providing the air-way is patent. The necessity

for always having a gas machine present during spinal anesthesia is thus demonstrated. Pharyngeal air-ways and an intratracheal set with laryngoscope and catheters should also be at hand if needed.

The position of the patient is of importance. Even though at least a 5° Trendelenburg position should be used during spinal anesthesia, when collapse occurs it should be changed to a 20° angle. This uses the force of gravity to aid circulation to the head and vital centers. Great caution should be exercised in avoiding the mechanical factors which may hamper respiration.

This small series of cases is illustrative of the type in which death would have ensued in several minutes if the anoxia had been allowed to persist. Most deaths under spinal anesthesia are of this type, the patient going into collapse, losing consciousness and dying. If when collapse occurs and consciousness is lost, prompt therapy is instituted, the coma can be reversed, and the patient recover, with no sequelae. In some cases the recovery occurs in a short time, 15 to 45 minutes, and in other more severe episodes of anoxia it may be delayed for up to 24 or 36 hours.

SUMMARY

The occurrence of coma during spinal anesthesia is a sign of severe anoxia of the cerebral centers. If this anoxia is allowed to persist for more than several minutes, death ensues, but when it is recognized as such as soon as it occurs and the proper physiologic therapy instituted immediately, the condition can be remedied and the patient saved. In some instances the recovery is preceded by a period of coma lasting up to 24 hours.

The therapy consists of prompt administration of oxygen, vasopressor drugs when necessary, Trendelenburg position, patency of the air-way and intravenous fluids. In order to be able to carry out this regimen the following precautions should be observed:

(1) A competent anesthetist should always be present and in charge of the patient's vital functions.
(2) An anesthesia machine should be in the room.
(3) A tray of stimulant drugs and syringe at hand.
(4) Pharyngeal airways and endotracheal equipment at hand.
(5) Use of Trendelenburg position.
(6) An infusion in major cases to provide a patent vein in case of peripheral collapse.

If these precautions are observed and immediate corrective measures taken when trouble does occur, the diagnosis of death from collapse of the patient under spinal anesthesia will rarely be made.

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